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THE SURGICAL TREATMENT OF PANCREATITIS[1]

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INTRODUCTION

DIGESTION, absorption, and assimilation of the body are primary functions and were accomplished facts before there was a nervous system, and the necessary control was obtained from certain organic internal secretions.

The sympathetic nervous system, probably mesoblastic in origin, came next as an aid to internal secretions and is still found closely connected with the organs which produce internal secretions. The thyroid is closely associated with the cervical sympathetic, the adrenals were for a long time supposed to be sympathetic ganglia, and the pituitary body is half sympathetic ganglia and half gland.

The development of the cerebrospinal nervous system was a later evolution which has become predominant. Nature has, however, not allowed the central nervous system to wholly usurp these original methods of control, hence internal secretions still act as "chemical messengers," and to a large extent they control digestion and assimilation. The struggle for supremacy of the advancing central nervous system over the internal secretions and the sympathetic ganglia gives rise to irregular function and may lie back of much of the prevalent neurasthenia, digestive neurosis, visceral ptosis, etc.

The surgeon cannot intelligently operate upon organs of double function without a full knowledge of their internal as well as their external secretions, for herein may lie the cause of the failure of a mechanically well executed operation to cure the patient.

The pancreas is an organ of internal and external secretion. Its internal secretion is actively associated with those of the intestinal glands, especially Brunner's glands in the duodenum, and also with the thyroid and adrenals. This activity is probably expressed through the islands of Langerhans, which seem to have a large influence on carbohydrate metabolism.

The external secretion of pancreatic juice is even more closely associated with that of bile from the liver, and so important is this latter association that by a process of evolution the pancreatic and common bile ducts have become joined just before entering the duodenum, and a small cavity, the ampulla of Vater, results, which acts as a mixing chamber.

A study of the comparative embryology of the derivatives of the primitive foregut, from which the stomach and duodenum to the common duct and the pancreas and liver are formed, throws an interesting sidelight on this anatomical relationship. The fœtal pancreas has two lobes, each with a duct having a separate opening into the duodenum. In the adult, the upper or duct of Santorini, although it has an orifice uncomplicated with the common mon bile duct, has become more or less obsolete, while the duct of Wirsung is the main excretory channel. This suppression of the direct flow of pancreatic juice through the duct of San-

[1] Read before the Mississippi Valley Medical Society, Louisville, Kentucky, October 14, 1908. For discussion, see p. 701.

torini in favor of the indirect duct of Wirsung, shows the importance of the association of the liver secretion to digestive function. The variability of the useful possibilities of the duct of Santorini indicates that this change is a recent evolution.

The liability of the appendix to disease, the wisdom teeth to early decay, and the little toes to corns and callosities, are examples of defects from progressive loss of function. Advancing function also makes for anatomical imperfections shown in the variability in the length, shape, and size of the sigmoid (Finney), as this derivative of the primitive hindgut increases its capacity as a fecal storehouse, and may result in constipation and irregular bowel action. It would appear that the changing digestive function is acting in the same way. The duct association of the pancreas and liver with its pathological tendencies shows the instability of short heredity. The gall-bladder changing from a bile storehouse to an organ needed for the relief of tension in the common duct and for the production of mucus, exhibits in gall-stone formation the same tendency to disease.

The lack of harmony in the results of experimental work on the internal secretions of the pancreas shows that this function is on the wane, just as happened in an opposite way in the thyroid, now an organ of internal secretion only, but at one time possessing an external secretion which escaped by way of the thyroglossal duct at the foramen cæcum at the base of the tongue.

The testicle is another example of internal and external secretion. The interstitial cells of Leydig which largely atrophy at puberty, through internal secretion continue the growth of the external evidences of manhood in those cases of sterility in which the testicles are incapable of producing semen. The association of normal function is here much the same as in the subject under consideration. The prostate adds its less important but necessary secretion to the testicular, much as bile is added to the pancreatic juice. Possibly the prevalence of prostatic disease may in its primary origin be the result of evolutionary changes.

The importance of the pancreas is shown by its protected situation, but it has, however, the fatal defect already pointed out of union of terminal facilities with the common bile duct, to which unfortunate mechanical arrangement most of the diseases of the pancreas are due, as it is thereby exposed to infections and diseases originating in the liver, gall-bladder, and ducts. We are also beginning to understand that the liver is equally unfortunate, as it is in the same manner exposed to diseases originating in the pancreas.

Our knowledge of pancreatitis may be said to be the result of an inquiry into the causation of some of the complications of gall-stone disease.

This lengthy preamble is rather by way of explanation of our lack of definite surgical knowledge, and to indicate that the following is but an imperfect clinical attempt to present the present status of a growing subject which must in a short time be subjected to more exact methods of study. (Fig. 1.)

ACUTE PANCREATITIS

The surgical treatment of acute fulminating pancreatitis presents difficulties that in many cases are unsurmountable. The patient, often a fleshy alcoholic male, exhibits all the symptoms of an acute perforation of some viscus, and when first seen is in a state of more or less collapse and a bad subject for an operation of any kind. The large majority of patients die whether operated on or not, yet immediate operation offers the best prospect of cure, and while the mortality is exceedingly high, it is not prohibitive. Of 59 cases reported by Robson, 23 recovered.

If the patient is seen during the first 48 hours, the abdomen should be opened in the median line above the umbilicus. More or less bloody serum will be encountered, and often, disseminated nodules of fat necrosis in the tissues even at this early time. The greatly swollen pancreas, having a semifluctuating "feel" showing gross hæmorrhagic infiltration, will easily be detected and should be incised in several situations, as in the successful cases of Mikulicz, Robson, and others. If the hæmorrhage from the incisions in the pancreas is severe, it may be necessary to pack with gauze and hold firmly in place with catgut sutures.

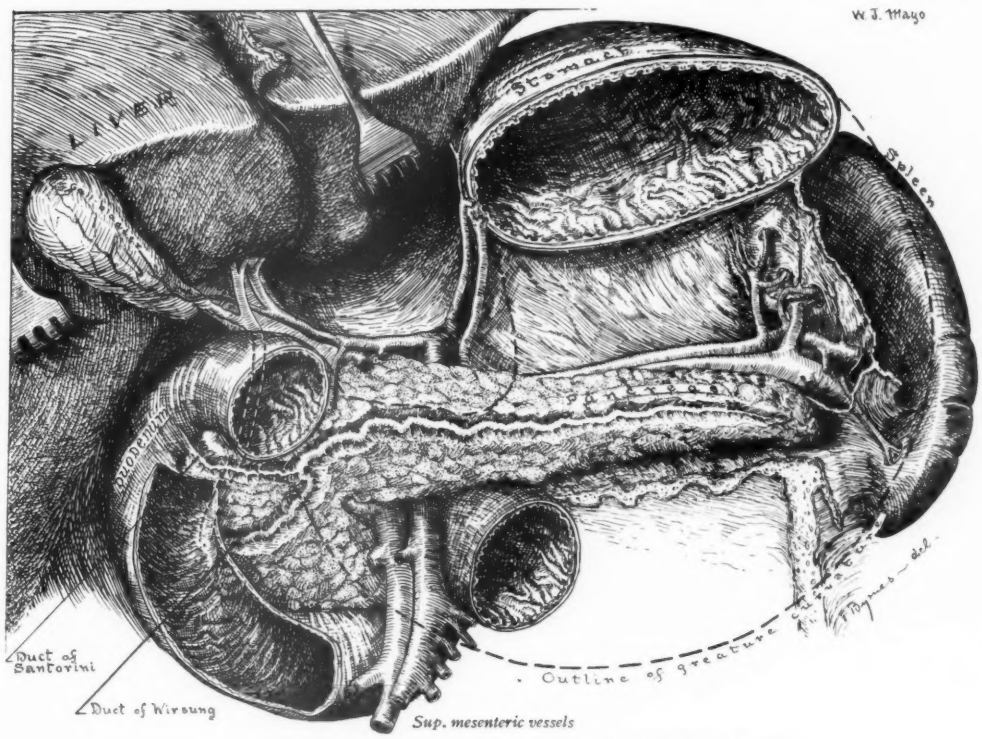

Fig. 2. Showing relation of pancreas to stomach, duodenum, and liver.

Free drainage should be furnished, and as a rule, through the anterior abdominal incision rather than through the posterior. The drains should be brought to the surface through split-rubber or glass tubes. As pointed out by Woolsey, the mere opening of the peritoneal cavity with drainage if fluids are present, seems to have a beneficial effect in some cases, evidently stimulating the natural resistance.

Should there be stones in the gall-bladder or ducts, or an acute infection of the biliary passages, removal of the stones and bile drainage through a right lateral incision should be done provided the patient's condition will permit. If any degree of jaundice is present bile drainage is essential.

After 48 hours the problem becomes more complex and depends largely upon the amount and virulence of the ensuing infection. As a rule, anterior drainage will be found more practical than posterior because the head of the pancreas, which is most often involved, lies in front of the spinal column and does not offer, anatomically, a good opportunity for the posterior route, although it must be admitted that some danger of infection is avoided if the phlegmon can be drained from behind by an incision outside of the erector spinæ muscles. However, free drainage anterior is capable of better management, and should more or less of the pancreas slough, which has happened in a number of reported cases, including one of our own, the after treatment can be more carefully carried out from in front, and the necrosed portion of the gland more readily removed than from behind.

SUBACUTE PANCREATITIS

The surgical treatment of subacute pancreatitis presents a more favorable field for operation. The patient has lived through the serious stage of acute infection, and his resis-

tance has been greatly increased. Our early knowledge of acute pancreatitis was based largely upon dead-house observations and we had an exaggerated idea of its fatality. For instance, fat necrosis, the result of the leakage of pancreatic ferments, is by no means so deadly a condition as has been supposed. We have operated upon a number of cases in which this result of acute pancreatitis was found, in which the patient had gone through a most serious illness and was apparently on the road to recovery, or at least was in the convalescent stage. On opening the abdomen, scattered all through the omentum, mesentery, and other fatty tissues, will be found these little split pea-like areas of fat necrosis. The peculiar disposition of these manifestations of the escape of fat-splitting ferments both behind and inside of the peritoneal cavity, is explained by the fact that in the fœtal state the pancreas is an intraperitoneal organ which by evolution has become extraperitoneal.

In subacute pancreatitis the pancreas may show evidence of localized septic accumulations, if so, these pockets should be opened and drained. If calculi are present in the gall-bladder or bile ducts, they should be removed and free drainage instituted. Especially should we be sure that no gall-stones remain hidden in the common duct under the enlarged head of the pancreas.

We have three times found localized non-infected accumulations of blood in the pancreas or adjacent tissue, the result of pancreatic apoplexia. All of these cases recovered after simple drainage. (Fig. 2.)

CHRONIC PANCREATITIS

Chronic pancreatitis was first described by Reidel in 1896, but the first case intelligently operated upon was one by Robson in 1900, and to him we owe the modern conception of this disease. The fundamental principle of the surgical treatment consists in temporary or permanent disassociation of the pancreatic and liver ducts by relieving the common duct of as much of its bile-carrying function as possible. In about two-thirds of cases, the common duct passes through the head of the pancreas to reach the duodenum and one-third, behind it. It will be readily seen that in two-thirds of the cases of pancreatitis, drainage through the common duct will be seriously interfered with and jaundice may be expected. In one-third, jaundice will not necessarily be present, but even here the common duct will usually be found more or less dilated. Dilatation of the common duct may occur quite independent of gall-stone disease, and both the ducts and the gall-bladder may be considerably distended without noticeable jaundice.

As pointed out by Courvoisier, 84 per cent of cases of stone in the common duct have a contracted gall-bladder due to the large amounts of connective tissue in its walls, the result of gall-stone irritation. Therefore, the size of the gall-bladder in chronic pancreatitis depends largely upon whether gall-stones are or have been present.

It is the rule that primary jaundice with the presence of a distended gall-bladder indicates malignant disease, but an exception must be made of these not infrequent cases of pancreatitis which do not have their origin in gall-stones, and, therefore, may have a distended gall-bladder. In malignant disease, the glands in the fissure of the liver are usually involved, early causing pressure upon the portal vein, one result of which may be the presence of free fluid in the abdomen, which is not a frequent result of chronic pancreatitis.

It can readily be seen that when the pancreas, as the result of infection, becomes swollen and causes pressure upon the common duct, that it seriously interferes with biliary drainage, and, though the pancreas may have become involved secondarily, it prevents that free drainage of the liver secretions which is so essential to the relief of the infection upon which the pancreatitis depends. The surgical treatment of chronic pancreatitis is not usually directed to the pancreas itself unless pancreatic calculi or other foreign bodies be present, but rather to the biliary tract, and is best accomplished by diverting the bile to the surface by means of a cholecystostomy, or to a new point in the gastrointestinal canal by cholecystenterostomy.

In the large majority of cases, gall-stones in the gall-bladder or biliary tract are the direct cause of the chronic pancreatitis. Up to September, 1908, in 2,611 operations upon the gall-

bladder and bile tract (C. H. & W. J. Mayo) the pancreas was coincidentally involved 200 times, or 7.6 per cent. In 325 operations on the common and hepatic ducts, the pancreas was involved in 22 per cent.

When gall-stones are present the removal of these stones with temporary free drainage by means of a cholecystostomy can usually be depended upon to cure chronic pancreatitis. The infection caused by the stones is readily relieved by this drainage, and in a comparatively short time the pancreatic pressure upon the common duct disappears and a permanent cure ensues.

In those cases of chronic pancreatitis in which no stones are present, cholecystostomy cannot be relied upon, because the continuation of the pancreatic disease lies outside of and beyond the biliary tract, and in these cases, more prolonged if not permanent diversion of the bile from its pancreatic association is necessary and cholecystenterostomy is indicated.

In several of our cases in which unsuspected chronic pancreatitis existed in connection with gall-stone disease, and in which cholecystectomy had been made, serious difficulties followed and were relieved only by secondary drainage of the common duct, all of which would have been avoided if in the original operation the gall-bladder had not been removed. For this reason, unless there is malignant disease in the gall-bladder, or this organ had lost its function through permanent obstruction of the cystic duct, we do not remove it if we have reason to believe that the pancreas is involved. A previous history of cholangitis, frequent attacks of jaundice, or stones in the common duct, contraindicate cholecystectomy, and the preservation of the gall-bladder enables a secondary cholecystenterostomy if necessary.

In a number of instances in which we had an opportunity to examine the common duct some time after cholecystectomy, we found that it was usually dilated, and in five of these cases, stones had formed in the common and hepatic ducts subsequent to the removal of the gall-bladder.

From the observations referred to above, it would seem that the gall-bladder has some considerable function. The amount of mucus which escapes from gall-bladder fistula when there is obstruction of the cystic duct goes to show that production of mucus is part of its function. Flexner showed that bile which contained considerable quantities of mucus was much less liable to cause pancreatitis than pure bile.

The fact that after the removal of the gall-bladder the common duct is so frequently found dilated must be taken into consideration, and it seems probable that the gall-bladder has at least two functions: first, to produce mucus, and second, to relieve common duct tension in the head of the pancreas. Both of these functions are evidently of great importance in preventing pancreatic complications.

The long accepted opinion that the function of the gall-bladder was wholly to store bile, seems improbable when we consider that from 20 to 30 ounces of bile are secreted each day, and the normal gall-bladder has a capacity of less than an ounce.

I am impressed with the fact that chronic pancreatitis is not only a much more frequent malady than has been supposed, but a more important one. In looking back over considerable experience in surgery of the gall-bladder and bile tract I find that a number of our cases that failed to make a good recovery, failed because of pancreatic complications. It is certain that much larger proportion of cases, especially those with a distended gall-bladder and a dilated common duct with or without stones, should be treated by a cholecystenterostomy than has been the practice among American surgeons.

Cholecystenterostomy was first performed by Winniwarter in 1880, but much of the early experimental work in connection with this subject was made by two justly celebrated Americans, the late Claudius H. Mastin, of Mobile, and the late W. E. B. Davis of Birmingham, Alabama.

In the performance of this operation, union has been made between the gall-bladder and four distinct parts of the gastrointestinal tract: the stomach, the duodenum, the jejunum, and the hepatic flexure of the colon. It must be conceded at the outset that union between the gall-bladder and the duodenum as near the orifice of the common duct as possible is the proper method, and should be practiced

where possible. Unfortunately in a considerable percentage of cases the duodenum will be found deeply placed and firmly held by adhesions, so that union between it and the gall bladder is a matter of great difficulty. Mobilization of the upper duodenum, as advocated by Kocher, is sometimes of value. It consists of a longitudinal incision through the peritoneum an inch or more in length just to the right of the duodenum in front of the kidney. Through this opening the duodenum is loosened from its posterior attachments and brought forward. By use of the curved clamps, union between the gall-bladder and duodenum can often be brought about, even when the parts are deeply situated, but in a fair number an anastomosis cannot be obtained without undue traction and some other part of the gastro-intestinal canal must be chosen.

We quite agree with Mr. Robson and Mr. Moynihan that if union cannot be safely effected with the duodenum, that a loop of upper jejunum should be selected. For this purpose the transverse colon is pulled upward and to the right until the jejunum can be seen at its origin just at the left of the vertebral column. A loop from two to three feet in length is formed that can be easily brought up over the transverse colon to the gall-bladder.

Mikulicz, in doing this operation, advised that an enteroanastomosis should be performed between the two limbs of the attached jejunum, and Monprofit has advocated a still more complicated method in which the jejunum is cut off, the proximal end being anastomosed to the gall-bladder and the distal end united to the side of the jejunal loop eight inches below, much after the manner of the Roux gastroenterostomy. Nothing in our experience has yet arisen to make these complicated procedures necessary, or desirable where the gall-bladder has been joined to a jejunal loop. It would seem wise to do enteroanastomosis as a secondary rather than a primary operation in those cases in which subsequent trouble did appear.

Union between the gall-bladder and the stomach has been advocated and practiced by Kehr in some cases. It is certainly an easy operation and, if it can be shown that the free drainage of bile into the stomach is not harmful, it would have many advantages from a technical standpoint. The stomach is thick walled, has a large blood supply, and a certainty of union would be assured. In four cases, we have found a spontaneous opening between the gall-bladder and stomach as the result of gall-stone sloughing. In these cases, operation proved that nearly if not quite all of the bile was being discharged into the stomach, apparently without trouble.

Union between the gall-bladder and the colon, theroretically at least, is very unfavorable. The function of the bile in the small intestines is lost and its stimulating effect upon the pancreatic juices is of very great importance. The fluids, however, and undoubtedly some of the solids, are saved to the economy through anastomosis with the colon, so that the patient is less thirsty and probably has stools more nearly normal than in those cases where the bile is carried permanently to the surface of the body by a cholecystostomy. In the few cases in which we made union between the gall-bladder and the colon no difficulty has arisen. Two of these patients have maintained excellent health for a number of years, and in neither did infection of the biliary tract result, a very serious danger, however, which threatens the patient whose gall-bladder has been united to the colon, teeming as it is with virulent organisms. It is probable that the continuous flow of bile furnishes mechanical protection. Fortunately circumstances demanding union between the gall-bladder and the colon will rarely occur.

As to the methods of accomplishing the anastomosis, but three are worthy of consideration: The suture which we have practiced to the exclusion of all others for some years, using the ordinary two row method exactly as in gastroenterostomy, making the opening from one to one and one-fourth inches in length. We prefer to use catgut for the inner row and linen for the outer row. If linen or silk is used for the inner row, it may hang for months before it sloughs its way into the intestinal tract.

In all of our early operations we used the Murphy button without any failures or misfortunes due to the button itself. If the button is used, it should be protected by four separated mattress sutures of linen uniting the sero-mus-

cular coats of the intestine and gall-bladder. A continuous suture of silk or linen should not be applied outside of the button as that may prevent it from loosening and dropping into the intestinal canal.

The McGraw ligature has also been used with good results, the method of its application being quite similar to its use in gastroenterostomy.

What shall we do in those cases in which the gall-bladder has been removed or is too contracted for the purpose of cholecystenterostomy? Drainage of the common bile duct will probably be necessary, but Moynihan and Robson report cases cured by breaking up adhesions about the biliary ducts and pancreas, while Eloeser has stretched the common duct, breaking up adhesions about it in the head of the pancreas with cure. The curious effect of the peritoneal incision in bringing about a cure in some cases, is difficult to explain, but that it has at times a certain usefulness cannot be denied.

In conclusion I would draw your attention to some rather rare conditions due to congenital anomalies of the pancreas in which small accessory lobes are found entirely separated from the pancreas itself, and provided with ducts opening into the viscus to which it is attached. These accessory pancreatic lobules are not infrequently found in connection with the stomach or duodenum, and occasionally with the jejunum or ileum. When they occupy these anomalous situations they are even more liable to inflammatory disease than the pancreas itself, and may cause strictures or diverticuli which give rise to marked symptoms. At some future time I hope to report a small number of such cases that have come under our personal observation.

THE ETIOLOGY AND PATHOGENESIS OF PANCREATITIS[1]

By WILLIAM D. HAGGARD, M. D., Nashville, Tennessee

INFLAMMATORY diseases of the pancreas have been the most inscrutable of all disorders of the great organs until their relationship with gall-stone disease offered a simple and rational explanation.

The important function of the gland is betokened by its sequestered and fortified position. It is less likely to intrinsic disease than any digestive organ. The only weak point in its defenses is the use of the duodenal opening in common with the secretory duct of the liver. In 1896 Reidel (1) called attention to the association of the tumor of chronic interstitial pancreatitis in the head of the gland with gall-stones and reported three cases. Robson had operated and cured such a case in 1892 by cholecystostomy and brought the subject strongly before the profession in 1900 (2). These are the cases that in the past have been deemed malignant on account of the indurated head of the pancreas and pronounced incurable, but the incidental removal of the offending gall-stones not only cured them but established the rational pathogenesis. Increasing experience has strengthened the correctness and importance of the relationship of gall-stones, especially in the common duct and the chronic interstitial form of pancreatitis. Robson has since found the ratio of pancreatic implication in these circumstances to be 60 per cent (20). Mayo's cases of common-duct stone (3) showed 18.6 per cent, but only the very obviously enlarged and unmistakably indurated instances were recorded; whereas, out of 168 operations for pancreatic disease he found 81 per cent were due to or accompanied by gall-stones somewhere in the biliary passages. Quenu and Duval (4) compute the simultaneous occurrence of gall-stones with chronic pancreatitis as being nearly 50 per cent. Williams and Bush (5) in a collection of 83 cases found gall-stones recorded as present in nearly 40 per cent. Egdahl (6) in an analysis of 105 cases, says about 42 per cent of the cases of acute pancreatitis are asso-

[1] Read before the Mississippi Valley Medical Association, Louisville, Kentucky, October 14, 1908. For discussion, see p. 701.

ciated with gall-stones, and considers them the most common single cause.

The most striking proof in the establishment of this relationship was the discovery by Opie, in a post-mortem of Osler's case, operated by Bloodgood, of a pea-sized gall-stone exactly occluding the ampulla of Vater, thus forming a continuous passage through which the bile traveled from the common duct directly into the pancreatic duct. To this carefully worked out and epoch-marking case he added the report of 7 others, and inferred that in many cases the stone may be temporarily lodged long enough to allow damage to the pancreas, and then escape, leaving no other evidence of its impaction (7). In one-half of the bodies examined by Opie the duct of Santorini is so contracted or obliterated that it cannot take up the work of the duct of Wirsung, when the latter is obstructed as it may be by the ordinary passage of a moderate-sized gall-stone through the pancreatic portion of the common duct without impaction at the ampulla. It is to be understood that the actual lodgment and retrojection of bile occurs with relative infrequence, as the conditions necessary for the exact mechanism are unusual. The stone must be small and round, "the diverticulum of Vater capacious, with a length at least greater than the diameter of its duodenal orifice." The essential conditions would therefore not allow of this often and where the duct of Santorini is larger than the Wirsungian, as it is in 10 per cent of cadavers, there would be little injection and no stagnation.

In addition to the mechanism described by Opie, Williams and Busch undertook experimental and anatomical investigations which seem to show "that some cases of acute pancreatitis may be caused by regurgitation of duodenal contents into the diverticulum of Vater, the way having been opened by the passage of gall-stones." It is therefore the anatomical peculiarities of the ducts and their variations which explain the supervention of pancreatitis in some cases of gall-stones and not in others.

The lower end of the common-duct is embraced by the head of the pancreas in nearly two-thirds of the cases. Stones sojourning in this portion will exert pressure on the pancreas and cause partial if not complete obstruction to its secretion, which may easily be infected either from the duodenum along Santorini's duct or through the Vaterian aperture. Inasmuch as the bile becomes infected in practically all cases of common-duct stone it is very rational to predicate the infections of greater or lesser severity of the pancreatic secretion from the same cause. Flexner has established that when the bile is greatly diluted with the admixture of mucus and injected into the pancreatic duct, only a mild interstitial pancreatitis will result, but when undiluted an acute and perhaps phlegmonous type will develop. It is on this ground that Robson makes a plea for the retention of the gall-bladder in operations upon it, whenever possible, in order that it may perform its normal function of diluting the bile with its mucus.

It will be seen that in many instances the etiology of pancreatitis is one with cholecystitis, and in the absence of gall-stones, acute septic infection must be regarded as the chief exciting cause, whether the organisms enter from the duodenum, or by way of the blood or lymphatics.

The bactericidal property of the pancreatic juice is assigned by Truehart as a reason why the pancreas is less often infected than the gall-bladder. The organism most commonly found in Egdahl's investigation of 105 cases was the *B. coli communis*; and then the streptococci and the staphylococci. The absence of bile from the duodenum as the result of obstruction seems to favor the virulence of the intestinal flora and the invasion of the duct of Wirsung by the infectious material (8). Korte demonstrated that injections of colon bacilli or fecal matter caused extensive interstitial changes (9). Pawlow proved that obstruction of the pancreatic duct would produce prancreatitis, from ligation of the duct in rabbits (10).

A small quantity of bile injected into the pancreatic duct caused death in 24 hours in a dog. Hlava and Flexner have reproduced by the injection into the duct of Wirsung of an artificial gastric juice and a dilute solution of Hcl, the picture of hemorrhagic and necrotic pancreatitis plus fat necrosis. Pancreatitis has also been said to be produced by pressure of tumors on the duct, duodenal stenosis from ulceration; from pancreatic calculi, intestinal worms, pieces of hydatid membrane in the duct, etc.

Aside from gall-stones gastro-intestinal disorders constitute the next largest etiological group. According to Egdahl they are responsible for nearly one-third of the cases. In nearly one-half of this number the gastro-duodenitis was associated with alcoholism. Only 8 out of 30 cases tabulated by Opie used it to excess and in 3 of these there were biliary or pancreatic calculi. Swelling of the duodenal mucosa, closing the ampulla of Vater, is the mechanical explanation. Pancreatitis from duodenal catarrh is probably the most common cause of what is generally called "catarrhal jaundice." The head of the swollen pancreas compresses the choledochus. An ascending pancreatic catarrh with stoppage of the secretion predisposes to calculi or calcareous deposit in the duct and is called by Robson "pancreolithic catarrh" (11).

Pancreatic lithiasis causes interlobular pancreatitis, and advancing to fibrosis may finally destroy the gland.

Maugaret (12) in an elaborate monograph of experimental and clinical data, just published, arrives at the following conclusions:

1. "Pancreatitis co-existing with cholelithiasis is not an affection independent from the latter, nor is it a simultaneous parallel disease, due to the same initial cause, and perpetuated by it. It is a true complication of cholelithiasis, as indicated by clinical observation and the results of treatment.

2. Its starting point is not an intestinal infection — neither directly nor indirectly, — the existence of which has never been clearly demonstrated.

3. The infection does not reach the pancreas by the canalicular route; this manner of propagation — which is not proved by the topography of the lesions, or by their histological localization — being hardly likely to occur, in view of the active bactericidal properties of the pancreatic juice, and in the normal physiological hurry of an excretory system stagnation is guarded against by the absence of a diverticular reservoir.

4. Neither is the pancreas reached by the infection through simple contiguity; this mode of propagation becoming probable only under conditions which obtain in very exceptional cases alone.

5. In all the cases of lithiasis, whatever they may be, the pancreatitis appears to be referable to a single cause, always the same in all these cases.

6. The unique cause of the pancreatitis in all the cases of lithiasis is the only constant condition which all these cases have in common, viz., infection of the gall-bladder. This is illustrated by the treatment of the pancreatitis, which consists essentially in the disinfection, or removal of the gall-bladder.

7. The gall-bladder infection is propagated to the pancreas by the efferent lymphatics of the gall-bladder, terminating at the important group of ganglia, both peri- and juxtaglandular, which occupy the region of the head of the pancreas; this group on the other hand receives the efferent lymphatics of the pancreas itself, which form extensive anastomoses at the surface. The organ is accordingly placed in lymphatic cross-roads, as it were, and is easily invaded by way of the periphery, as soon as the infection has the tendency to pass beyond the ganglionic boundary.

8. It appears accordingly that among the four possible modes of infection of the pancreas — hematogenous, canalicular, by contiguity, and lymphatic — the last named is not only a possible route, but a common one."

The causative factor next in frequency to gall-stones and duodenitis is mumps, which occurred in about 10 per cent of Egdahl's collective investigation.

The complication of mumps is of course metastatic. The specific cause being unknown, the complication is likewise obscure.

Gordon Sharp (13) reports the case of a boy with pancreatitis, and no parotitis 10 days after his sister had mumps. He passed blood and fat in the stools. Another case of pancreatitis preceded mumps. He concludes that "(1) Pancreastitis might precede parotitis in one and the same patient and (2) one patient in a household or in an infected area might show parotitis while another might only have pancreatitis, both due to one and the same causal agent."

Cuche, in one epidemic, observed 20 cases of pancreatitis out of 26 cases of mumps.

Edgecomb (14) saw 5 instances of this metastasis in 33 cases occurring epidemically in a

boys' school. All presented acetone and diaceti acid in the urine.

They nearly all recover, but Lemoine and Lehasset observed one fatal case. They are characterized by epigastric pain increased on pressure, nausea, vomiting, local swelling, diarrhea, slight fever, no glycosuria, but have jaundice and hematemesis.

Sidney Phillips (15) reports a case of pancreatitis with jaundice and acute parotitis in scarlet fever as being uncommon and as a probable explanation of the jaundice occasionally occurring in scarlatina. It supports the view that "catarrhal jaundice" is more often due to obstruction of the common duct by pressure of the enlarged head of the pancreas than to swelling of the duodenal mucosa only.

There have been enumerated among other causes: Influenza and other zymotic diseases, syphilis, tuberculosis, malaria, typhoid fever, scarlet fever, gastric and duodenal ulcer, carcinoma, boils, emboli, retrojection of bile from unknown causes, and traumatism. Arteriosclerosis is also mentioned by Martina as causative (17).

Thrombosis of the smaller vessels of the abdominal viscera from malaria, has been demonstrated by Machiafava and Barker. Chalmers Watson (18) found jaundice and tenderness over the pancreas with a positive Cammidge reaction in a malignant type of malaria.

Following typhoid only a few cases have been reported, one shortly after (Chauffard and Revant) and another remote (Moynihan). It could occur from duodenal extension or by metastasis. When we consider the great incidence of typhoid and the comparative frequency of gall-bladder infection and parotitis in typhoid, it seems curious that the pancreas is not oftener attacked. It seems to show that its bacteriiodal resistance is good, when there is no mechanical interference with its defense and drainage.

Pancreatitis may occur from slight injuries. Jones' patient was seized with severe epigastric pain on turning in bed. The pancreas may be torn by a fall without other evidence of injury. "An auto-destructive ferment, which dissolves the walls of the blood vessels and produces hæmorrhage is set free by any injury to the pancreas however it may be inflicted." Robson. (19).

The distinctive and rather constant pathologic change in acute pancreatitis is disseminated fat necrosis. The cause is the diffusion of the pancreatic juice and its ferments into the omental mesenteric and subcutaneous fat, splitting up the neutral fat into its component fatty acid and glycerine. The latter is absorbed and the former unites with the lime salts to form yellowish white non-elevated, opaque spots about the size of a millet seed that look like droplets of candle-grease. The process is non-bacterial. As supporting this view Oestrich (20) reports a case of fat necrosis in which the pancreas was entirely free from necrosis, but the liver was necrosed and he believes the pancreatic juice was carried there by a blood current in omental adhesions to the liver.

Sawyer obtained sterile cultures from the necrotic patches, and Flexner showed the presence of the fat-splitting ferment in these areas. When micro-organisms are present they are secondary invaders.

Cases attended with fat necrosis are not necessarily fatal. Robson and Mayo have reported successful operations in its presence.

Berard and Chalier (21) on reporting a fatal case consider that death is due to the action of trypsin upon the solar plexus; or to an intoxication of the organism by massive absorption of the trypsin poured in abundance into the abdominal cavity. This is the view accepted by Gulecke, Lenormant, and Lecene.

Eloesser (22) asserts that fat necrosis with hæmorrhagic pancreatitis is dependent upon some lesion of the pancreatic tissue, and caused by some ferment, the exact character and mode of action of which has eluded all types of research. Stagnation of the pancreatic juice alone will not produce it until it is "activated."

The duct of Wirsung may be doubly ligated and divided but will not produce this disease unless in so doing the cells of the gland parenchyma are injured, allowing the escape of this kinasis. This activating principle may also be liberated by ischemic necrosis of the damaged gland-cells; or it may be formed by the bile when retrojected into the Wirsungian duct in the presence of obstruction at the papilla of Vater; or it may be inoculated by the intestinal juice regurgitating into the pancreatic duct.

The hæmolytic or eroding property of this ferment combined with the pancreatic secretion causes a hæmorrhagic extravasation, which damages new portions of the parenchyma, thus liberating more of the ferment - like substance to cause further hemorrhage and fat necrosis. This is characterized externally by collapse and the first clinical signs of the disease.

CONCLUSIONS

1. Gall-stones are etiologically associated with pancreatitis in from nearly one-half to over three-fourths of the cases (43 per cent of Egdahl to 82 per cent of Mayo).

2. Anatomical peculiarities and variations of the terminal pancreatic ducts are the determining factors in the occurrence of pancreatitis in some cases of gall-stones and not in others.

3. Lodgment of a stone in the ampulla of Vater temporarily or permanently causing retrojection of bile into the pancreatic duct (Opie) causes acute hæmorrhagic pancreatitis.

4. The chronic indurative or interstitial type is caused by stones lodged, resident, or escaping through the common duct.

5. Bacterial invasion is secondary to some unphysiologic or mechanical internal injury.

6. Infection of the pancreas by the lymphatics in cases of cholecystitis is probably a more frequent avenue than by continuity.

7. Gastro-duodenitis appears to be responsible for about one-third of the cases.

8. "Catarrhal jaundice" is probably caused by inflammatory swelling of the head of the pancreas compressing the common duct of the liver, in many instances.

9. Mumps causes about 10 per cent of the cases of pancreatitis, and some other infections may likewise cause it by metastasis.

10. Fat necrosis with acute hæmorrhagic pancreatitis is caused by some injury to the gland cells, and access of bile or enterokinase which activates the hæmolytic property of pancreatic juice.

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PHYSIOLOGY AND CHEMICAL PATHOLOGY OF THE PANCREAS IN PANCREATITIS[1]

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PANCREATITIS in any form has not been a subject for common discussion until within recent years. Our knowledge of the grave and acute forms of the disease has been derived from lessons taught in the post mortem room, or there has been laid bare upon the operating table the "pancreatic drama."

Now we have learned that inflammation of the pancreas, especially the chronic form, is by no means of rare occurrence. Although this latter form of the disease has been encountered most frequently during operations upon adjacent organs, it must be granted that it possesses its own clinical interest.

Inflammation of an organ leads to disturbance of its function. The detection of such disturbance of function is not infrequently the earliest and only key to the diagnosis, if not, indeed, the only clinical manifestation of the disease.

Of the normal pancreas we know that it possesses at least two functions — the secretion of ferments for the digestion of foods in the small intestine, and the secretion of a substance or substances, that have an important influence upon the assimilation of food, particularly of the sugars, in the tissues. It has also been demonstrated, that when an inflammatory disease of the pancreas runs a course sufficiently acute so as to leave definite end-results, and yet permit of accurate clinical observation, that the external, as well as the internal, secretions are abolished, so that on the one hand intestinal digestion fails, and on the other hand metabolic disturbances arise that lead to sugar waste.

This statement sets forth briefly what definite knowledge we have of the pathologic physiology of the acute and subacute forms of pancreatitis. From a clinical aspect the acute form runs a course so violent that delay for the purpose of extensive investigation of functional disturbance, in furtherance of a clinical diagnosis, is usually not indicated.

The chronic forms of pancreatitis offer better opportunities for clinical investigation, and I may say that in the nature of things we are wholly restricted to clinical observation. Chronic pancreatitis, as an experimental disease in animals, does not yield opportunities for observations that have clinical application. This statement does not apply to certain chemical problems connected with the disease, to which I have made reference elsewhere.[1] Against the clinical observations it must be urged, that the results have only a comparative and relative value, because the conditions for an accurate scientific investigation are rarely present.

In the acute form of pancreatitis there is usually a rapid destruction of the entire parenchyma of the gland; chronic pancreatitis is characterized chiefly by a hyperplasia of the fibrous tissue between the lobules and between the acini, giving rise to the interlobular and the interacinous forms of the disease. As a result we have degeneration and destruction of the glandular elements. In this connection it has been noted that the islands of Langerhans are less readily destroyed than the secreting cells of the acini. The former are perhaps not at all involved in the interlobular form of pancreatitis.

If the cause of the chronic form of pancreatitis is external, that is, if it is due to the invasion by bacteria, or to the passing of foreign substances through the duct of Wirsung, the cells of the acini must suffer directly and quickly. From analogous conditions in other organs we may infer that this leads to an early disturbance in their function. The existence of such an insufficiency is borne out by the clinical observation where a chronic pancreatitis presumably exists. There be-

[1] The Journal A. M. A., September 5, 1908.

[1] Read as part of a symposium on Pancreatitis at the thirty-fourth annual meeting of the Mississippi Valley Medical Association, in Louisville, October 13-15, 1908. For discussion, see p. 701.

comes evident an indigestion of the intestinal contents, so that large quantities of undigested and unaltered fat and undigested muscle fibres are passed in the fæces. The digestion of starches is not subject to control.

Such apparent indigestion is not conclusive evidence of an actual pancreatic insufficiency: the pancreas may secrete in a normal manner, while the duct of Wirsung may be blocked, as not infrequently happens, by a gall-stone in the common duct or in the ampulla of Vater, inducing sooner or later, however, a chronic pancreatitis. Again, the absence of bile, enteritis, gastric insufficiency and a faulty test diet may give rise to intestinal indigestion simulating pancreatic insufficiency. There may also exist a pancreatitis without giving gross evidence of intestinal indigestion. The gland has a fairly large secreting area which may only be partly involved, thus not giving rise to an actual secretory insufficiency. Moreover, the action of the secreted ferments is quantitatively such, that even a reduced amount may meet the digestive requirements.

It has been suggested that in the very early stages of chronic pancreatitis from external causes, there may take place an oversecretion of the pancreatic juice, giving rise to intestinal disorders.

The internal secretion does not seem seriously disturbed in chronic pancreatitis; at least evidence of it, such as glycosuria, is not frequently observed. I am satisfied from my own observation, that an alimentary glycosuria can be more readily induced in patients suffering from chronic pancreatitis, and this I believe to be due to injury to the islands of Langerhans.

Certains forms of disease of the liver must, of course, be excluded.

The manifestations of pancreatitis that are subject to chemical investigation, have been considered collectively as the chemical pathology of the disease. Here again one is tempted to speak of the difficulties which are met with in such studies, because the influence of organs other than the pancreas cannot be excluded. However, such difficulties do not deprive of interest the immense amount of work done upon the subject.

Intimately associated with the more recent work done in this direction is the name of Cammidge, of London.

In 1904 Cammidge published the results of his studies on the urine of patients suffering with pancreatic disease. He set forth that he had isolated from such urine a substance which he believed to be fairly characteristic of pancreatitis, and to which he ascribed great diagnostic importance.

The work of Cammidge was carefully repeated by various investigators, and his conclusions were shown to be unfounded.[1] Cammidge then modified his methods of investigation, and made a further contribution to the subject in 1907, wherein he described his improved, or "C" reaction. He maintains that there occurs in the urine of patients having an inflammatory disease of the pancreas a substance, which on being boiled with strong hydrochloric acid will split off, or yield a body having the chemical character of the sugars; free sugar, however, being absent.

In an examination of 87 cases of disease of the pancreas, he found the reaction to be positive in 71, or in 82 per cent. Of 117 supposedly non-pancreatic cases, 113 were negative, and four positive in reaction. Fifty specimens of normal urine were all negative. Cammidge could not establish the identity of the substance in question, but in one instance he found it to be pentose.

The present contention of Cammidge derives support from investigations that have since then been made by other investigators.

Thus Eichler, of the University of Berlin, produced experimentally an acute pancreatitis in two dogs, and he obtained a positive reaction according to the method of Cammidge in each case.

Eloesser reported from the University clinic at Heidelberg sixteen cases, in which a positive reaction was found upon an examination of the urine, and which at operation showed a pancreatitis.

Edgecombe, of London, reported a positive reaction in a case of parotitis. Cammidge himself made the test.

Watson reported a number of cases. He reported a positive reaction in all cases of

[1] For the literature up to date, applying to the Cammidge test, see *Schroeder*, Urinary Manifestations in Disease of the Pancreas, The Journal A. M. A., September 5, 1908.

acute and chronic pancreatitis, and a slightly or moderately positive reaction in some other forms of disease.

From personal observation I may say that the observations published by Cammidge in 1904 as the so-called "A" and "B" reaction, are without value. During the past ten months I have done considerable work with the improved or "C" reaction, and I believe that the observations thereby made are not only interesting from the standpoint of pathology, but of value as a diagnostic factor.

At the last meeting of the American Medical Association I presented the result of a study of the urine of 85 individual cases of various disorders, pancreatic and otherwise. In all, 110 chemical analyses were made. Of these 85 cases, 60 patients had disease of the digestive organs, or were complaining of digestive disorders. In 11 of these 60 cases a positive reaction was obtained. Thus, of four cases, in which a clinical diagnosis of chronic pancreatitis had been made, three were positive and one negative. One case of cancer of the pancreas was positive in reaction; it was the most pronounced reaction I have ever seen. Three cases of cancer of the stomach were examined: two gave a positive reaction, and one was negative. The two positive cases were operated upon, and the pancreas was found to be involved in each case. I had the opportunity of making a post-mortem examination in the case which gave a negative reaction, and the pancreas was found not to be involved. One case of abscess of the pancreas was examined for the Cammidge reaction three days after operation and gave a negative reaction.

Of four cases of gall-stones that came to operation, one, having a stone in the common duct, gave a positive reaction, and one similar case a negative reaction. One case in which the gall-bladder was packed with stones, gave a negative reaction. At the operation the pancreas was found to be hard and nodular. Of four cases of catarrhal jaundice, one gave a positive reaction. Two cases of tumor of the upper abdomen, presumably of pancreatic origin, gave a positive reaction. One case of tuberculosis of the peritoneum was positive.

The urine in 59 cases that gave no evidence of pancreatic disease, gave a uniformly negative reaction.

It was impossible to obtain in any of the positive cases enough of the substance to identify its character. In the case of cancer of the pancreas reported, I was enabled to determine the melting point of the phenylhydrazin precipitate — 180° C. This corresponds closely with the melting point obtained in the animal experiments of Eichler — 182 degrees.

Aside from the occurrence of fat necrosis, glycosuria, and the pancreatic reaction of Cammidge, the acid intoxications and the elimination of a large quantity of calcium oxalate crystals in the urine have been suggested as being especially associated with pancreatitis, but that has not been proven. Of great interest is the observation to which attention has been called by Robson and Cammidge, namely, that the coagulability of the blood is reduced in the presence of inflammatory disease of the pancreas, thus giving rise to a tendency to hæmorrhages even in the absence of jaundice. The withdrawal of calcium salts from the system has been suggested as a direct cause of this condition.

In making the Cammidge test I have closely followed the directions laid down by the author. The slight modification that I have finally adopted serves, to my mind, to facilitate the work.

The method that I have found convenient is as follows: 40 c.c. of the urine, filtered, acid reaction, free from albumin and sugar, are boiled with 2 c.c. of strong hydrochloric acid for ten minutes. After partly cooling, 8.0 gm. of lead carbonate are gradually added. When the reaction is complete, the mixture is chilled, filtered, and 10 c.c. of filtrate diluted to 20 c.c. with distilled water. This solution is precipitated with 2.0 gm. of tribasic lead acetate, filtered, and the excess of lead in solution removed by the addition of 2.0 gm. of sodium sulphate, bringing the mixture to a boil, chilling, and filtering to 20 c. c. To this filtrate there is now added a mixture of phenylhydrazin hydrochlorate 0.80 gm., sodium acetate 2.0 gm. and of 50 per cent glacial acetic acid 1 c.c., and the whole is boiled on a sand bath for ten minutes. The solution is

then filtered while hot, and set aside to crystallize. The precipitation of crystals, sometimes only in microscopic quantities, of the characteristic formation of ozazones, makes a positive reaction.

The following precautions must be observed:

1. The urine must not contain albumin or sugar, and must be fresh and filtered clear.

2. Positive reactions must be verified by making the phenylhydrazin test for free sugar in the urine, using the same method without previous boiling. Fehling's test is not sufficiently delicate. The sufficiency of removing sugar by previous fermentation has not been proved.

3. Tribasic lead acetate ($Pb(C_2H_3O_2)_2 . 2PbO$) must be employed. The solution of lead subacetate of the U. S. Pharmacopeia is not satisfactory. I have employed it side by side with the tribasic lead acetate and have obtained misleading results with the former. Before realizing this difference I had some correspondence with Cammidge on the subject, and I agree with him that the tribasic lead is the only suitable precipitant, because of the uncertain and indefinite composition of the pharmacopeial preparation. The positive reaction may be marked or may require microscopic examination. I have noted precipitates that were not crystalline and, therefore, negative. Precipitation may take place immediately on filtering or may occur after standing twenty-four hours. Except as to size, the crystals that I have observed in various positive reactions were apparently of the same system. I can not concede any value whatever to such tests as solubility in sulphuric acid, as tending to point to the nature of the disease, and I have, therefore, never applied this test.

THE DIAGNOSIS OF PANCREATITIS[1]

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UNTIL the beginning of the present century, the clinical diagnosis of pancreas disease was limited to recognition of traumatic injuries, pancreatic cysts more especially, and malignant growths.

At the present time we may add to these conditions a fairly positive clinical diagnosis of acute and chronic pancreatitis in at least a portion of the cases which come under our care suffering from these conditions.

Pancreatitis, necrosis of the pancreas, and hæmorrhagic conditions were recognized at times incidentally during the progress of abdominal operations, especially those performed for the removal of gall-stones. At autopsy it was usually difficult to determine to what extent the gland had been digested by its own ferments, consequently the diagnosis of diseases connected with the pancreas was badly neglected in a general way until the brilliant work of Mayo-Robson (1) brought this organ to the proper attention of the surgical profession. Later the same author in connection with Cammidge (2) brought out a most ingenious test which in the hands of its author and a few others seems to make a laboratory diagnosis of pancreatitis quite possible. In 1905, my assistant, Dr. J. L. Yates, a thoroughly trained laboratory expert who had also great experience in clinical surgery and surgical pathology, applied the Cammidge test most carefully to a large number of surgical cases in our care in the Augustana Hospital. The study proved extremely fascinating but did not lead to practical results. This was probably due to the fact that we demanded evidence not requiring a personal element.

We were however convinced that the method contained merit and should be encouraged.

[1] Read before the Mississippi Valley Medical Society, at Louisville, Ky., October 14, 1908. For discussion, see p. 701.

Since that time a few investigators have had results corresponding to those of Cammidge, which have been most brilliant in the estimation of such keen observers in the surgical profession as Robson and Moynihan. Most interesting of these are the observations of Cloesser (12) which are set forth in a scholarly review of 16 cases in a paper of more than 200 cases.

Other observers have obtained positive reaction in many cases in which the pancreas was proved to be normal at subsequent operation, while a negative reaction was found in some cases in which chronic pancreatitis was later demonstrated to be present.

From these facts it seems plain that further development is necessary before the test can be generally employed but that the method should not be discarded, because it certainly contains scientific value, especially in the hands of investigators trained in the observations of most delicate chemical reactions.

So large a proportion of patients suffering from gall-stones or from an infection of the gall-bladder and ducts also suffer from pancreatitis that the general symptoms of cholecystitis should make one suspicious of the probable presence of pancreatitis. The proportion of cases in which pancreatitis complicates cholecystitis has not been positively determined. Various competent authorities place it at from 6 per cent to 30 per cent. The statistics of Mayo (14) and those of Robson are the most complete on this subject at the present time.

It has seemed to me that if in cases of cholecystitis there is a distinct point of tenderness upon pressure at Mayo-Robson's point half way between the end of the ninth rib and the umbilicus and a second area of tenderness upon deep pressure for a distance of five to ten centimeters from a point a little to the right of the umbilicus upward over the right rectus abdominus muscle, we may safely make a diagnosis of pancreatitis in all cases in which duodenal ulcer can be reasonably excluded. Two symptoms of duodenal ulcer must be absent,— (a) hyperchlorhydria with eructations, and (b) pain before meals when the stomach is empty.

Another common symptom which is present in cases of pancreatitis is the pain radiating to the midscapular or the left scapular region. Robson and Moynihan (3). This referred pain has been looked upon as indicative of gastric ulcer but this seems true only when there is also pain upon deep pressure at a point half way between the ensiform appendix of the sternum and the umbilicus.

In making a diagnosis of pancreatitis complicating cholecystitis we must, of course, bear in mind the facility with which secondary infection of the pancreas is likely to occur in cases in which there has been long continued infection of the bile tracts. Hence if one operates early in cases suffering from cholelithiasis or cholecystitis one must find a smaller proportion of pancreatic complications than if one operates late.

Aside from the Cammidge test a number of authors have published chemical tests for diagnosis which seem worthy of further study. Solomon (10) claims that in giving von Noorden's oatmeal diet, patients suffering from pancreatitis have typical butter stools. He also states that an abnormally large amount of licethin is found in stools of patients suffering from pancreatitis. When they are placed on an egg diet from 0.4 to 1.2 gm. will be excreted in 24 hours while under the same conditions if the pancreas is normal never more than 0.1 gm. is excreted.

Lepine (11) has made extremely interesting experiments which are too complicated to be discussed here but which merit careful study by those especially interested in this subject. Schlecht (13) describes Mueller's test determining the presence or absence of pancreatic activity. This test is so simple and convenient that it must prove valuable if future tests will establish its absolute reliability.

The patient is given a test meal and two hours later a calomel purge. A few drops of the stool are sterilized by heating to a high temperature and then placed on an Agar plate containing Loeffer's serum. This material is kept at a temperature of 131° to 141° F. for twenty-four hours. If trypsin is present the serum shows pronounced depressions where this substance has been active, if absent the serum remains smooth.

ACUTE PANCREATITIS

It is extremely difficult to make a positive diagnosis of acute pancreatitis, because the attack differs in no way from several other violent acute conditions, like perforation of gall-bladder, duodenum, or the posterior wall of the pyloric end of the stomach. Renal colic, ectopic gestation, appendicitis, and acute intestinal obstruction have also been mistaken for this condition and more commonly the reverse has been the case. In acute pancreatitis there is terrific pain in the right upper quadrant of the abdomen, the patient is severely shocked, nausea and vomiting is usually present. The abdominal muscles are extremely tense. Tympanitis develops rapidly but is more marked in the upper right quadrant. Sugar is sometimes present in the urine. Sappington (4). Fitz (5) in a splendid article written nearly twenty years ago laid especial stress upon a previous history of occasional attacks of indigestion before the development of the above violent symptoms. In cases in which there has been chronic pancreatitis for a considerable period (6) he directed our attention to the fact that fat is visible in fæces upon simple inspection.

Moynihan (7) has found the muscles not rigid in these cases, which is contrary to my own observation, but as no individual observer sees a large number of these cases, it will be interesting to give especial attention to this symptom in the future. He also directs attention to the steady rising of the pulse from the onset. There is quite generally an increase of temperature which rises very high in the very acute cases.

Elliott (8) directs attention to the fact that in acute pancreatitis if a tumor mass can be felt the surface is tympanitic on percussion because of the location of the gland behind the duodenum and pyloric end of the stomach.

Opie (9) has observed cyanosis as a symptom, which I too have noticed in two cases of acute pancreatitis. He is of the opinion that the rise in temperature is probably due to secondary changes and not to the acute pancreatitis itself.

STATISTICS

Should one attempt to formulate conclusions relating clinical diagnosis of pancreatitis upon the available statistics at the present time, these conclusions would surely be hopelessly unreliable because there has been as yet no uniformity of observation.

Egdahl has carefully studied the histories of 107 cases of acute pancreatitis which he has been able to collect from the literature. His paper is full of interest and will serve to encourage more accurate observation but it will require many years of continued study of a very large number of cases before it will be possible to make practical use of statistics in this disease to aid materially in diagnosis.

With all of these observations before us it seems reasonable to demand at least a probable diagnosis in a considerable proportion of cases of acute pancreatitis. If we add to this the practical fact that this condition as well as all of the conditions with which it is likely to be confounded require surgical treatment, the absence of an absolutely positive diagnosis ceases to be so serious a matter as it might otherwise be considered.

The same conditions obtain in connection with chronic pancreatitis and consequently it is quite as important for the patient that the surgeon should not overlook the presence of acute or chronic pancreatitis when the abdomen has been opened as it is to make a clinical diagnosis before the operation.

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PANCREATIC DIABETES AND ITS RELATION TO GALL-STONES[1]

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An inquiry into the peculiar interrelationship recognized to exist between gall-stone disease and pancreatic diabetes, aside from its purely pathological interest, is important clinically chiefly for three reasons, viz.:

1. Because cases of persistent, usually very intractable, glycosuria occur that run their course under the guise of an "idiopathic" diabetes mellitus, but that are in reality due to gall-stone factors.

2. Because cases occur that present all the essential clinical features of cholelithiasis, accompanied by continuous or intermittent glycosuria, that are in reality cases of chronic pancreatitis and not at all of gall-stone disease.

3. Because the two disorders occur together much more often than is generally assumed and produce a complicated disease-picture that is highly confusing and very commonly misinterpreted.

IDENTIFICATION OF DIABETES AS PANCREATIC

The identification of a glycosuria as due to pancreatic diabetes is usually very uncertain because it implies the notoriously hazardous diagnosis of pancreatitis. Further difficulties arise when gall-stone disease or other hepatic involvement, as is so often the case, coexist; for hepatic insufficiency, due to any cause, *per se* often produces an alimentary glycosuria that may be genetically quite independent of pancreatic disease. If, clinically, similar conditions obtained as in experimental ablation of the pancreas, the diagnosis of pancreatic diabetes would be exceedingly simple. Cases presenting such a syndrome must however be considered great rarities; they occur only in very advanced stages of pancreatic disease with complete obliteration of the glandular parenchyma, and not always then.

DIFFICULTY OF DIAGNOSIS IN PANCREATIC DISEASE

The diagnosis of pancreatitis is rendered particularly difficult for the following reasons:

The close proximity of the pancreas to liver, stomach, duodenum, the intimacy of the vascular, lymphatic, nervous supply between these organs, render it apparent that symptoms emanating from the pancreas can very well be referred to adjacent organs and explain why disorders about the liver, stomach, duodenum so often actually precede, accompany, or follow pancreatic disease.

The enlargement of the head of the pancreas, the most constant and most striking gross manifestation of pancreatic disorders, moreover, causes pressure upon blood vessels, bile channels, nerve trunks, thus producing a great variety of remote symptoms, the primary pancreatic origin of which very well may and usually does escape detection.

Again, it must be remembered, that the *whole* glandular parenchyma of the pancreas is rarely involved so that on account of the different functions exercised by the various glandular areas of the organ only one or the other of these functions may be perverted or suppressed. When one considers finally that none of the different pancreatic functions are exclusively specific to the gland but that each can, to some extent at least, be vicariously assumed by other organs of the body, then the enormous difficulty of making the diagnosis of pancreatic disease either from general symptoms or from specific symptoms of pancreatic debility must be recognized.

AMBIGUITY OF GENERAL SYMPTOMS

The general symptoms never suffice alone to make a diagnosis. There is nothing typical about the pain, nothing about its location, intensity, or character that indicates the pancreas and the pancreas alone; generally, in fact, the pain simulates very closely the distress in cholelithiasis, the paroxysms of gall-stone colic, even the radiations into the subscapular and lumbar regions and the location of the most tender pressure point very near the gall-bladder region. There is nothing about the digestive disorders — the nausea, belching, vomiting

[1] Read in a Symposium on Pancreatic Disease before the Mississippi Valley Medical Association in Louisville, Kentucky, October 14, 1908.
For discussion, see p. 701.

(often of bile-stained material), the diarrhœa, the constipation — that might not be found in many gastrointestinal disorders. There is not infrequently icterus, some irregular fever of no particular type or occasionally subnormal temperature. In advanced cases of chronic pancreatitis there is generally much emaciation, a profound anæmia with oligocythemia, oligochromemia and degenerative signs about the erythrocytes resembling closely the picture of a primary pernicious anæmia. Sometimes the skin becomes bronzed. Two general symptoms that point with a little more directness to the pancreas remain to be mentioned, viz., a peculiar disarrangement of the fat distribution about the body, leading to the formation of rolls of adipose tissue especially about the front and side of the chest and the abdomen, resembling somewhat the picture of Dercum's disease (adiposis dolorosa); and profuse salivation (sialorrhœa pancreatica), possibly due to reflex stimulation of the salivary glands from the diseased pancreas (may be a vicarious hypersecretion of amylolytic material).

SPECIFIC FUNCTIONAL TESTS

More definite information can, as a rule, be gleaned from a study of those phenomena that are attributable to inadequacy or debility or some of the pancreatic functions; often these symptoms can be brought out with especial clearness if special functional tests are made to determine the power of the pancreas to properly disassimilate the albumins, the fats, the carbohydrates in the intestinal canal. In obstruction of the pancreatic duct, and, not so regularly, in far reaching destruction of glandular parenchyma the stools, are bulky, because so much pabulum fails to be disassimilated at all and hence escapes absorption. There is much fat in the stools and the muscle nuclei pass through the intestine in an undigested form (Schmidt's test); in addition lipase (Hewlett's test) may appear in the urine, also pentose (Cammidge reaction), and much dextrose.

THE STEATORRHŒA

The steatorrhœa, however, is by no means a constant phenomenon, for the emulsification, splitting, saponification, of the fats can be vicariously assumed in part, at least, by the gastric juice, the bile, and intestinal bacteria; hence in early stages of pancreatic disease marked disturbances of the fat assimilation are really quite rare and often absent. The sign is really only of distinct diagnostic value if very large quantities of fat appear in the stools without the existence at the same time of icterus, acholic stools, or severe intestinal disturbances; for in the presence of either or all of the latter symptoms the steatorrhœa may very well be due, not to pancreatic disease, but to disease of the bile passages. Sometimes the production of an alimentary steatorrhœa by giving a fat test meal (50 c.c. of olive oil) after two or three days of an ordinary mixed diet is of value, for a normal subject should absorb the bulk of these 50 c.c. of oil; in pancreatic disease most of this fat reappears in the stools imparting a peculiar consistency to the fœces and causing the appearance of grayish white flakes and lumps in the stools; chemically, too, of course, a quantitative fat determination shows an increase above the normal. These fat stools differ from the fat stools of cholelithiasis by the appearance of the fat as neutral fat and the presence of bile pigments or their congeners.

THE NUCLEAR TEST

The appearance of large quantities of undigested muscle fibre is an altogether ambiguous symptom occurring in many disorders involving disturbances of the function of the small intestine. The appearance of undigested nuclei in the stools is indicative of pancreatic disease only provided there is no profuse diarrhœa causing the muscle fibre to be rushed through the intestine, and provided the muscle fibres are not allowed to remain in the bowel longer than 24 hours, for putrefactive processes, too, may cause disintegration of the nuclei.

THE GLYCOSURIA

Glycosuria, finally, is an altogether inconstant symptom. The pancreas, presumably by means of an internal secretion furnished by the cell groups of Langerhans, aids in the regulation of the carbohydrate metabolism. How, we do not know. Complete removal of the pancreas in animals invariably produces the complete syndrome of diabetes and one should expect the same in complete destruction of the

gland in man; there are, however, a number of cases of carcinoma of the pancreas and of advanced chronic pancreatitis on record with destruction of most of the acini and the islands of Langerhans, in which glycosuria either failed completely to make its appearance or appeared for a time *only to vanish again later on.* This seems to indicate quite clearly that, if enough time is given, the regulation of the carbohydrate metabolism can be assumed by other organs, notably, in all probability, by the glands of Brunner in the duodenum, that are genetically very closely related to the pancreas.

FREQUENCY OF PANCREATIC DIABETES

Hence, glycosuria, scil. pancreatic diabetes, is a much more common symptom in acute pancreatitis and at the onset of pancreatic disease, *especially when associated with cholelithiasis*, than in later stages. This is very important. In occlusion of the pancreatic duct (or experimental ligation) with damming back of the pancreatic secretion, glycosuria is rare, for the islands of Langerhans seem to resist this insult very well and only undergo degeneration when nearly all the other glandular elements of the pancreas are destroyed. It appears that in individuals with a diabetic heredity the occurrence of glycosuria is much more apt to take place in even slight pancreatic involvement. The administration of large quantities of starchy or sweet food may occasionally demonstrate the existence of a reduced tolerance for carbohydrates; therefore a special test for alimentary glycosuria should be made in every case presenting symptoms significant of pancreatic involvement.

The glycosuria, therefore, is a symptom of peculiar value in pancreatic disease; its presence being indicative of pancreatic involvement, its absence not necessarily invalidating this diagnosis.

PANCREATITIS SIMULATING CHOLELITHIASIS

Given a more or less vague and inconstant syndrome of the above type, simulating in many important aspects, as I have taken pains to point out, the common picture of cholelithiasis, it is not surprising that the latter diagnosis is so often arrived at when in reality the pancreas is, at least, originally at fault. It is due chiefly to failure to perform specific functional tests for pancreatic involvement, not once but several times, in all cases presenting the picture of cholelithiasis, especially when accompanied by glycosuria, that pancreatic involvement is so often overlooked, pancreatitis considered to be so rare, and the disease so commonly discovered only on the operating table or at autopsy. For this reason, too, surgeons were the first to recognize the frequency of the disorder and to forcibly call our attention to its importance. Hence, finally, many cases that are diagnosed at cholelithiasis and operated upon for this disease, *recover* from their symptoms notwithstanding the fact that the bile passages were found quite intact and gall-stones absent. These are often cases of unrecognized pancreatitis.

CHOLELITHIASIS AND PANCREATITIS

Aside from the cases of pancreatitis in which the syndrome of cholelithiasis is simulated, but in which gall-stone disease is not present, we have much more frequently a combination of gall-stones and diabetes in which the type of the diabetes should, properly, be specified as pancreatic. What has been said in the preceding paragraph in regard to the necessity of performing tests of the functional capacity of the pancreas when cholelithiasis is simulated, applies with particular emphasis to cases in which the presence of gall-stones is determined or rendered probable and that run their course accompanied by diabetes. For in all such cases the prognosis of the diabetes is as a rule much better and the treatment quite different than in the so-called ordinary idiopathic types of the disease.

In attempting to interpret the occurrence together of pancreatic diabetes and gall-stones three possibilities must be taken into consideration, viz.:

1. The presence of gall-stones in the gall-bladder or their passage through the bile passages into the duodenum may produce the pancreatic changes.

2. The disorder about the pancreas may be the cause of the gall-stone formation.

3. Both the gall-stones and the pancreatic diabetes may be due to some common cause acting simultaneously upon the liver and the pancreas.

The pathogenetic relationship of the two diseases has been fully discussed by the essayists who have preceded me; so that it is unnecessary to enter into further details myself. Gall-stone disease precedes the pancreatic disease in most cases and chronic pancreatitis must be considered a common sequel of cholelithiasis; here and there a pancreatitis by mechanical compression of the common duct passing through the head of the gland, or by secondary infection causes stasis of bile, infection of bile channels, cholelithiasis; now and then infection, enterogenous or hæmatogenous, lues, alcohol, trauma, cause both pancreatic and hepatic involvement.

THE TREATMENT

Medical treatment of pancreatic diabetes, especially when complicated with cholelithiasis, is exceedingly unsatisfactory. In fact, drinking cures are, as a rule, very poorly borne, although *after* operative treatment they are of great value and often materially aid in the production of recovery. Antiluetic treatment is sometimes of value. Pancreas preparations never do harm, but at best they are a substitution remedy that may palliate some of the most distressing derangements about the digestive tract, may aid a great deal in promoting better absorption of the food, hence better general nutrition, but never produce a cure.

In view of our inability in many cases to distinguish between gall-stone disease and pancreatitis, or to determine whether or not pancreatitis complicates gall-stones, or vice versa, an exploratory laparotomy is always indicated. The surgical standpoint has been fully discussed by others.

The good effects of a laparotomy in both acute and chronic cases of pancreatitis are often striking, the active hyperemia from the incision probably of itself acting beneficially, as in tuberculous peritonitis.

The removal of hepatic concretions very generally exercises a beneficial effect upon the pancreatitis and the glycosuria. Removal of pancreatic concretions, the better drainage of the abdominal cavity (ascites, fat necrosis), are all useful and necessary steps. The presence of pancreatic diabetes in these cases does not counterindicate laparotomy.

A STUDY OF THE COMPARATIVE IRRITATIVE PROPERTIES OF SOME MERCURY COMBINATIONS USED AS ANTISEPTICS

By GEORGE B. LAWSON, A. M., M. D., Roanoke, Virginia

THE mercury salts have held and still hold a very prominent place in the line of chemical disinfectants, especially the bichloride of mercury. Their common use has caused them to be combined for various reasons with other chemicals; the most common of which are ammonium chloride, hydrochloric acid, tartaric acid, and citric acid. Probably the most common combination is with ammonium chloride and this is probably the worst, giving precipitates with the hard waters. One can usually tell the ammonium chloride combination in dilute solution by the milky precipitate, by the scum, and discoloration of vessels which contain the solutions. The scum and precipitate is partly avoided by first mixing the strong bichloride of mercury and ammonium chloride solution with sodium chloride. This probably forms a double salt of sodium and mercury chloride.

One of the popular forms in the New York hospitals is glycerin bichloride of mercury and ammonium chloride. The bichloride of mercury may be concentrated in this solution as high as 1-8. This is a much quicker way of making dilutions than using the tablets. However, it is a poor combination on account of the precipitation. This may be partly avoided by mixing with sodium chloride before dilution.

Because of this precipitation, the acid com-

binations with bichloride were brought out. These in dilute solutions in hard water will stand for months without scum and without clouding or precipitation. They will also not precipitate with albumin until all the acid is used up to form the acid albuminate.

Yet the most interesting point is which combination has the most bactericidal power. Laplace[1] brought out this point especially when the bacteria were combined with serum. He showed that the bactericidal power of bichloride of mercury was four times as great when combined with acids.

However, for disinfecting hands, open wounds, etc., a very important point is how strong may the antiseptic be used without injuring the skin and the wounded surfaces. This point seems to be brought out very little in the use of antiseptics. By study of the effects of the various combinations of the mercury salts, we have endeavored to classify their irritant effect on the skin.

Thus similar parts of the body, in most cases the ankles, were scrubbed with green soap, then washed with alcohol and ether so as to remove superfluous dirt and fat. Our endeavor was to put the skin in about the same condition as for a surgical operation. Then on one ankle was put a cotton compress of a certain size into which was soaked a definite quantity of a definite solution of bichloride of mercury in distilled water. On the other ankle was put exactly the same preparation in regard to size of compress and amount of solution except that there was added a chemical whose action it was desired to study. Over these compresses were put rubber protective, cotton, and bandages. The compresses and solutions were replaced at intervals sufficiently frequent that the skin was always kept damp with the solution. The skin was observed at various times and as soon as a noticeable inflammation was produced, the compresses were removed while those on the other ankle were kept on sufficiently long as to produce a similar inflammation. By this method one can study very accurately the relative irritative properties of various chemicals on the human skin.

[1] Saure Sublimat. Losung als Desinficiendes mittel und ihre verwendung in Verband stoffen, von Ernest Laplace. Deutsche Medicinisc e Wochenschrift, October 6, 1887.

Our experiments are tabulated as follows:

1. Female, age 24, fair complexion. Bichloride of mercury, 1-500 on right; bichloride of mercury, 1-500 and tartaric acid 1-500 on left. These were removed after 19 hours, left ankle was about three times more inflamed than right.

2. Female, age 17, medium complexion. Bichloride of mercury, 1-500 on right; bichloride of mercury, 1-500 and tartaric acid 1-500 on left. These were removed after 24 hours. Left leg was more inflamed than right with scalding effect. Itching began on left side almost immediately after application.

3. Female, age 20, blonde. Bichloride of mercury, 1-500 on right; bichloride of mercury, 1-500 and tartaric on left. These were removed after 17 hours, the redness was much more marked on left.

4. Male, age 12, dark complexion. Bichloride of mercury, 1-1000 on right; bichloride of mercury, 1-1000 and tartaric 1-1000 on left. Eighteen hours later, left shows redness with papules. No effect on right. Eighty hours from beginning right is a little more affected than was left at 18 hours.

5. Male, age 14, medium complexion. Same experiment as above except that both legs had severe itching and both applications were removed at 28 hours. The left showed few reddened papules while right was normal.

6. Female, age 18, blonde. Bichloride of mercury, 1-1000 on right; bichloride of mercury, 1-1000 and tartaric 1-1000 on left. Twenty-four hours later, left is reddened with vesicles. Right leg is negative. Forty-eight hours later, right leg is still negative.

7. Male, age 28, light complexion. Bichloride of mercury, 1-1000 on right; bichloride of mercury, 1-1000 in 1% salt solution on left. Twenty-eight hours later, both legs were inflamed, but left not so much as right.

8. Male, age 24, dark complexion. Bichloride of mercury, 1-1000 on right; bichloride of mercury, 1-1000 in 1% salt solution on left. Ninety-six hours later, right leg was a little redder than left.

9. Male, age 26, dark complexion. Bichloride of mercury, 1-1000 on right; bichloride of mercury, 1-1000 in 1% salt solution on left. Seventy-two hours later, right leg was more inflamed than left.

10. Male, age 24, light complexion. Bichloride of mercury, 1-1000 on right; bichloride of mercury, 1-1000 and citric acid 1-1000 on left. Ninety-six hours later, left leg is reddened and inflamed. Right normal. Twenty-four hours later, right leg is not as inflamed as was left.

11. Male, age 28, light complexion. Bichloride of mercury, 1-1000 on right; bichloride of mercury, 1-1000 and citric acid 1-1000 on left. Seventy-two hours later, left leg is erythematous and shows vesicles while right is normal. Forty-eight hours later, right is not as red as was left.

12. Male, age 24, light complexion. Bichloride of mercury, 1-1000 on right; bichloride of mercury, 1-1000 and citric acid 1-1000 on left. 72 hours later left leg is inflammed and shows vesicles; 24 hours after

this right leg is about the same as was left on previous day.

13. Female, age 17, medium complexion. Bichloride of mercury, 1-1000 on right; mercuric iodide 1-1000 and potassium iodide 1-1000 on left. Four hours later, left leg shows marked redness with itching. Right is normal. Twenty-four hours later, right leg shows slight redness.

14. Female, age 18, light complexion. Bichloride of mercury, 1-1000 on right; mercuric iodide, 1-1000 and potassium iodide 1-1000 on left. Three hours later, marked burning on left side. One hour after this, left leg is markedly inflamed. Twenty-four hours after beginning, right leg was almost as inflamed as was left.

15. Female, age 22, dark complexion. Bichloride of mercury, 1-1000 on right; mercuric bromide, 1-1000 and potassium bromide 1-1000 on left. Fourteen hours later, left leg showed marked hyperæmia while right is only slightly reddened.

16. Female, age 22, light complexion. Bichloride of mercury, 1-1000 on right; mercuric bromide 1-1000 and potassium bromide 1-1000 on left. Five hours later, left leg is markedly inflamed. Twenty-four hours from beginning, right leg shows slight dermatitis.

17. Female, age 40, medium complexion. Bichloride of mercury, 1-1000 on right; mercuric bromide, 1-2000 and potassium bromide 1-2000 or left. Twenty-four hours later, right leg is a little more irritated than left.

18. Female, age 22, light complexion. Bichloride of mercury, 1-1000 on right; mercuric bromide, 1-2000 and potassium bromide, 1-2000 on left. Twenty-four hours later, left leg shows some irritation, while right is almost negative.

In using very strong solutions of bichloride of mercury, say 1-250 to 1-750, the hands take on a black, rough appearance which persists until the affected skin wears off. This happens also when the hands are used a great deal in 1-1000. By studying the effects of alternating the tartaric acid and bichloride with ammonium chloride and bichloride in a few cases in the dressing rooms, the tartaric acid combination did not seem to affect the hands more than the other combination.

The finger nails of certain persons have more of a tendency to turn black by the constant use of solution of bichloride of mercury than have those of other people. The nails are made blacker by sunlight. One must note the idiosyncrasy which exists in certain persons for bichloride on the skin, thus one case, a trained nurse with dark skin, had used on her hands 1-1000 bichloride for 15 minutes, this caused extensive dermatitis with exfoliation over part of hand and forearm. This patient stated beforehand that she had this idiosyncrasy.

In applying wet antiseptic dressings, one notes that the water tends to evaporate at the edges thus causing more concentrated solutions at those points, which brings about a local inflammation. One also notes that where antiseptic solution is applied repeatedly to dressings that the evaporation of water will presently bring about a very strong antiseptic solution in the dressing.

CONCLUSIONS

1. Mercuric chloride is more irritant to the skin when combined in solution with acids.

2. Mercuric bromide and iodide are more irritant to the skin than mercuric chloride.

3. Mercuric chloride is slightly less irritant to the skin when in solution in normal saline than when in distilled water.

SOME CLINICAL CONCLUSIONS AS TO THE VALUE OF VACCINE THERAPY IN SURGICAL INFECTIONS [1]

By JOHN C. HOLLISTER, M. D., Chicago

SOME two years ago a laboratory was established at St. Luke's Hospital, Chicago, by Dr. L. L. McArthur and myself, for the purpose of investigating the clinical value of vaccine therapy as based upon Wright's opsonic theories in a series of general surgical cases. The aim was to follow as closely and accurately as possible Wright's own laboratory technique, and the therapeutic efforts were, for the greater part of the time, based carefully upon the status of the opsonic index. Our desire was to ascertain if possible whether

[1] Paper read before the Mississippi Valley Medical Society at Louisville, Kentucky, October, 1908.

Wright's suggestions are really of value in the treatment of surgical infections.

The object of this paper is to give as briefly as possible a somewhat preliminary report of some facts and figures we have learned and some conclusions it seems justifiable to make.

The *Laboratory* facts and figures, dealing with the questions of "normal index," "reliability of the index," etc., etc., have appeared from time to time in articles written by the different members of the laboratory staff and will be touched upon here only in reference to one or two points.

In drawing conclusions every effort has been made to avoid any overstatement of facts. Every case reported has been watched and progress noted repeatedly by more than one investigator, the great majority by more than two. The statements given are not exaggerated. It has been appreciated how easy it is to assign all improvement to the specific therapy. We have tried to avoid this. Most of the cases treated over long periods of time were receiving continually the same general, surgical, dietetic, and hygienic treatment that the cases would have received if no vaccine had been given. In the following tables no case is reported as greatly improved unless it seemed evident to the different investigators that the improvement was due to the vaccine. A considerable number of cases that were already improving but were given vaccine have been entirely neglected in making up the figures. Only those cases are considered at all that have received the vaccine thoroughly and persistently, varying in time, of course, according to the infection.

STAPHYLOCOCCUS CASES

Some 60 cases were investigated in all. 34 *cases were persistently treated*, 12 of acne (face and neck); 15 of boils or large furuncles; 2 of empyema; 1 mixed infection with case of lupus; 2 fistula, mixed.

Of the 34 cases 13 were cured, 38 plus per cent; 11 were somewhat improved, 32 per cent; and 8 were not improved, 24 per cent.

The eight cases showing no improvement were 4 of acne, 3 of boils, 1 empyema (mixed lung tuberculosis. (25 of above cases were treated with autogenous, and the others with stock vaccine.) Of these 25 cases, 8 improved greatly, 32 per cent; 9 improved slightly, 36 per cent; making 68 per cent; 8 did not improve, 32 per cent.

Of the 9 cases treated by stock vaccine seven were cured and two were slightly improved.

For a considerable length of time, while very careful laboratory work was done, we gave the vaccines according to the opsonic index. After many hundred indices had been made and compared with one another it was determined to give the vaccine at stated intervals, regardless of the index, for reasons given below. This we have done on almost all of the recent cases.

CASES OF TUBERCULOSIS

163 cases in all were studied from both laboratory and clinical standpoints. Some 3,000 indices were determined. Forty-one cases were treated carefully over sufficient period of time to draw clinical conclusions. Eight of these were generalized infections, 33 localized. Of these 41 cases, 29 were bone and joint cases, of which six had also other evidences of tuberculosis.

Case	Improved		
	Much	Slight	Not
Bone and Joint, 29 cases.....	9	10	10
Genito-urinary tract, 2 cases....	2		
Pulmonary tuberculosis, 3 cases			3
Pulmonary tuberculosis plus bone and joint disease, 4 cases..... (also included in bone and joint above)			4
Glands of Neck, 4 cases		2	2
Generalized (genital tract, skin, lungs)	1		
Peritonitis.....			1

Of all these 41 cases 28 or 70 per cent improved (15 greatly, 13 slightly), 13 or 30 per cent were unimproved.

Ten of the 13 unimproved were of the generalized type. In five cases, all of which improved very much, passive hyperæmia was used in addition to the tuberculin. In 9 of the 15 greatly improved the beneficial effects of the vaccine were *very marked indeed.* In one case there can be no reasonable doubt that a life was saved (urinary tract infection with great emaciation, etc. See former reports).

GONOCOCCUS INFECTIONS

Until some nine months ago much difficulty was experienced in collecting a sufficient number of cases of gonorrhœa in which the true clinical status of the individuals could be carefully and persistently followed and the possibility of reinfection avoided. In fact although some 60 were studied and compared with 50 normals and 500 or more indices were made, we could be sure of but one clinical conclusion. This was that the vaccines are of no definite value in acute cases. Last spring we were extremely fortunate in having an opportunity of beginning investigations upon a series of 50 cases of gonorrhœa in young women between the ages of 14 and 20. This research work is steadily continuing and it might be well now to give a very brief general report of some impressions already gained. It is of course much too soon to draw any conclusions relative to prevention of complications, etc.

After bacteriological and physical examination of these 50 cases they were divided into five groups, each group resembling as nearly as possible the others with regard to pathological findings. Then to the first group was given the usual hospital treatment (ichythol, silver nitrate, etc.) that similar cases had received previously in the institution. The treatment of group 2 was that of the gonococcus vaccine, plus hygienic cleanliness. Group 3 was given the usual antiseptic treatment in addition to the vaccine. To group 4 was given nothing but irrigations and spongings of physiological salt solution. To group 5 has been given doses of antigonococcic serum so highly recommended by Dr. Rogers and Dr. Torrey of New York.

From time to time each case has been re-examined bacteriologically and physically by at least two, usually three, investigators. The final results of the investigations will be brought out in full some months later. The only conclusion we are at all justified in drawing as yet is that the gross discharge from the cervix, vagina, and urinary meatus diminished more quickly in the group receiving vaccine therapy plus the usual hospital antiseptic treatment, and those receiving vaccine alone, and this is as true of the latter as of the former.

A large number of cases of other infections, such as colon bacillus, etc., justifying clinical conclusions, has not been observed, although of marked improvement in individual cases we are quite certain.

The following statement may be of interest as regards the value of the opsonic index, etc.:

We believe that the index is by no means an unfailing guide for determining the amount of the vaccine and the time of administration, for the following reasons: 1. Many cases showing persistent high index show no clinical improvement. 2. Fewer number of cases improve although index remains comparatively low. 3. Many cases continue to improve greatly when treated without regard to the index. These reasons apply especially to staphylococcus infection where we have found as a rule before treatment normal or high indices. We do believe there is a normal index. We *do* believe that the opsonic index, when obtained with careful technique, is a distinct aid in diagnosis, especially in the chronic localized infections. A persistently low or varying index is a very strong indication indeed that the patient is suffering from a specific infection. We cannot agree with Wright, though, in drawing conclusions from variations of but one or two tenths in the index. If the patient is receiving vaccines, either those elaborated in his own body as in systemic infections, or those administered with therapeutic intent, the index of that patient's serum seems to lose to a certain extent its reliability as a register of opsonic content. Evidently many other opsonin-like bodies are manufactured by the tissues in response to vaccination.

THE OPERATIVE TREATMENT OF UNILATERAL LUNG TUBERCULOSIS BY TOTAL MOBILIZATION OF THE CHEST WALL BY MEANS OF THORACOPLASTIC PLEURO-PNEUMOLYSIS

By PROFESSOR DR. P. L. FRIEDRICH
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Translated by Chas. M. Matter, M. D., Chicago, Illinois

MANY attempts during the past decades to treat tuberculosis of the lungs by surgical means have sufficiently shown the relative hopelessness of such procedures. Whether the caverns were treated by injections or opened by the knife or even resection of parts of the lung according to the distribution of the disease, the slow processes of healing after such attempts resulted more and more in excluding surgical means in treating tuberculosis of the lungs.

It is not necessary at present to go over the different experiments and their spare results. A new impulse to influence lung phthisis by surgical means was given by the work of Murphy, Forlanini, and Brauer. By producing an artificial pneumothorax they caused a more or less extensive compression of the lung, with reduction in size of the caverns and variably extensive exclusion of the diseased lung from the respiratory function. What has been accomplished in this field up to date and which may be found in the publications of Forlanini and Brauer cannot fail to impress one who follows the course of disease from an objective standpoint. It is here sufficient to indicate the work of these men.

Since Brauer has shown that adhesions between the layers of the pleuræ occur in one-fourth of the cases of tuberculosis of the lungs, making it manifestly impossible to introduce nitrogen between the pleuræ costalis and pulmonalis in order to produce a pneumothorax of a certain graded extent, the question arises of finding some other method that will cause a lessening of the lung volume and shrinking of the cavernous lung tissue. One would naturally think of thoracentesis, making an intercostal incision, loosening the adhesions between the lung and chest wall, then after healing of the wound repeating the pneumothorax injections.

I tried this procedure once on a patient referred to me by Professor Brauer and found certain difficulties in the way. The layers of the pleura remain thin in spite of existing adhesions and during their manual separation, without the aid of sight, one is apt to tear into the lung tissue. In consequence complications are introduced which can easily result in empyema pleuræ or mediastinitis and endanger the patient's life. In our case the pneumolysis did not succeed. A cavern lying near the pleura pulmonalis was torn open, and a mild serous pleural exudate, which was present, became an extensive pleural suppuration. The patient, who was already very poorly nourished and suffering from tuberculosis of the other lung and the intestines, died after several weeks. This case did not encourage us to repeat the method, in spite of its recommendation by others.

Moreover, in cases of extensive unilateral lung tuberculosis with formation of caverns, in which nothing could be accomplished by the pneumothorax method, we thought of the idea of Quincke which he published in 1888, recommending the local mobilization of the chest wall over areas of tubercular lung disease. Quincke later on referred back to this proposition without, however, being able to collect operative experiences with it, and specialists in diseases of the lungs, such as Karl Spengler, Turban, and Landerer, have earned recognition by their work in the local mobilization of the chest wall by means of the more or less extensive resection of several ribs, even though the results have not been definite.

I will not enter into an explanation of these preliminary experiments in this place, as I have exhaustively gone into this subject in several publications. (See Verhandlungen der deutschen Gesellschaft für Chirurgie, 1908; Langenbeck's Archiv. für Klinische Chirurgie,

Berichte des Aertztlichen Vereins zu Frankfort am Main, vom 1 Juni, in der Münchener Med. Wochenschrift; Sitzungs-Berichte der Gesellschaft zur Föderung der gesamten Naturwissenschaften zu Marburg, vom 8 Juli; Medicinische Klinik, 1908, No. 33. Moreover, in keeping with the desire expressed to me by the editors of this journal some time ago, I will now explain the method I use at present in extensively mobilizing the chest wall by means of total removal of bone with the most careful preservation of the pleura costalis.

Before going into an explanation of the operation I will first recapitulate some important clinical and pathologic-anatomic data. We know that in the cavernous phthisis of young individuals between 16 and 35–40 years, while the organism is living under favorable nutritional conditions, that the diseased lung possesses a great tendency to shrink, so marked that the opposite lung, heart, mediastinum, diaphragm, and clavicular fossa are drawn toward the shrinking lung. Only the bony chest wall is unable to follow suit and this appears to be the most important hindrance in the process of recovery. We know further from the autopsy table that the so-called lung phthisis of slow course shows changes in the walls of the caverns which cause them to resemble bronchiæctatic caverns. Further and not seldom, we see how massive fibrous connective tissue induration can affect the whole diseased lung with connective tissue isolation of and healing processes in areas of tubercular disease to such an extent as we relatively seldom see in other organs. We may therefore deduce the conclusion that our efforts must be directed toward supporting by all possible means this tendency of an extensively diseased lung to shrink. Now that we have seen that the bony chest wall is the chief obstacle to complete retraction it seems justifiable to attack cavernous phthisis by means of extensive rib resections. Only further observations on human beings can teach us if it is really possible to favorably influence or complete the process of healing.

Knowing as we do that the exact estimation of the extent of lung tuberculosis by the methods of physical diagnosis is often very difficult (Quincke v. a.) and that the Röntgen method while it is a great help in ascertaining the extent of phthisis-pulmonalis, does not at times give definite results, we can see that a restricted resection of the chest wall (removal of 3–5 ribs to an extent of 6–10–15–20 cm.) can only have a limited effect upon the shrinkage of the lung volume. In this regard our latest method differs from the earlier ones in that we can so alter the technique that an almost total removal of bone from the chest wall can be accomplished, thus diminishing the lung volume to $\frac{1}{2}-\frac{1}{4}$ of that which it was previously and at the same time causing a definite shrinking of the cavernous portions of the lung. It was also necessary that such a severe operation in one who is very ill, should be so evenly balanced in all its parts that the immediate operative results should not be behind the expectations, and to this end we have striven with all the means at our command.

After the explanation of the technique I will more clearly define the indications for the individual cases according to the experience of Prof. Brauer and myself.

I. PREPARATION OF THE PATIENT

Most of the patients here considered have fever, considerable expectoration, and differ a great deal in the amount of nourishment they take. We give no antipyretics or medicines to combat the expectoration, but use all the means at our command to increase the appetite and amount of ingesta. In case they have not been confined to bed for many weeks or months as the most of them are, we leave them in bed and exhaust all the details of the physical and X-ray examination.

On the day of the operation nothing is allowed per os, except occasionally a cup of tea early in the morning. Twenty minutes before the beginning of the operation, .015–.02 morph. mur. hypo. After this all possible expectoration is encouraged for a long time in order to free the bronchi and caverns of secretion and thereby diminish the danger of aspiration during the anæsthesia.

II. THE OPERATION

Patient is laid in the semi-oblique position with the body turned half over on the well side. The arm is held vertically up in the air. The

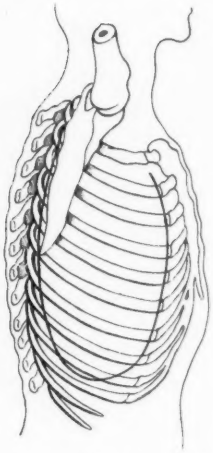


Fig. 1. Line of incision.

cleaning with soap, ether, and alcohol according to rigid aseptic principles over the whole chest. After this the incision is made similar to that used in Schede's method of thoræoplasty in empyema, the skin and muscle being best rendered analgesic by Schleich's infiltration method. Then a flap is formed by beginning the incision 3 fingerbreadths external to the edge of the sternum at the height of the 2d or 3d rib, downwards external to the nipple to the 10th rib, posteriorly up again to the 2d dorsal spine. Occasionally for cosmetic reasons, I have deviated from this line as in Fig. 6. In dividing the M. Serratus Ant. Maj., the ends of the serrations are allowed to remain, otherwise the whole muscle with vessels and nerves is rapidly displaced upward. The M. pect. Maj. and Latiss. Dorsi are deeply nicked and drawn strongly aside with retractors. Now the bony chest wall is exposed (time occupied so far is 3 min.). Then with the greatest rapidity the ribs are resected leaving periosteum and intercostal muscles behind, being however all the time careful not to puncture the pleura costalis, as a pneumothorax at the present stage would greatly endanger the patient, and as a marked increase in the secretions of the caverns would greatly promote the dangers of aspiration. This is very much more easily avoided by the use of the positive or negative air-pressure apparatus of Brauer

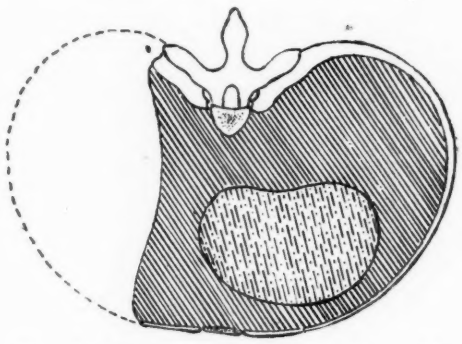


Fig. 2 (diagrammatic). Diminution in volume of lung by right-sided operation.

or Sauerbruch, but everything depends upon completing the operation without their help. After the detachment of the 2d to the 10th ribs from the anterior cartilages to the spine and especially after the hold of the 2d and 10th ribs gives way, the whole lung covered by the intact pleura costalis sinks back toward the hilus and in operations on the left side the heart can be seen rising above the level of the lung. By practice such rib resections as these removing in all 180–220 cm. of bone can be done in 25 min. from the time the bone removal is begun. After the removal of projecting pieces of intercostal muscles, nerves, and periosteum, and careful hæmostasis, the muscle flap is carefully approximated with 20–30 buried catgut sutures, the skin sutured with silk, and a thick drainage tube is inserted along the spine.

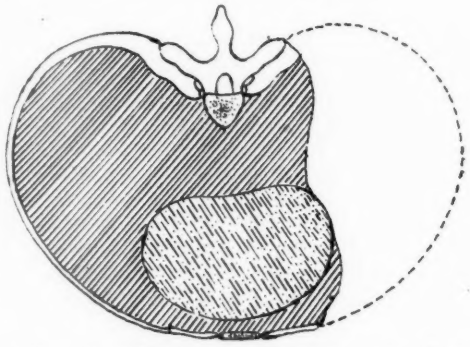
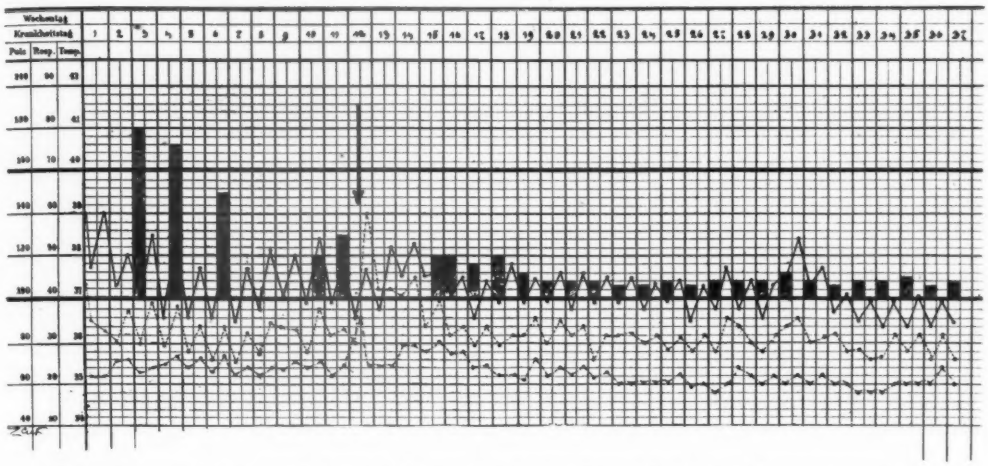


Fig. 3 (diagrammatic). Diminution in volume of lung by left-sided operation.



Temperature, straight lines. Pulse, dotted lines. Respiration, dot-and-dash lines. Sputum, gray columns. Curve of Case IV. Man, 31 year sold, whose pictures, ten weeks after operation, are given in Figs. 4 and 5.

As a rule, if all these conditions are carefully attended to during the operation the patients stand the operation all right and leave the operating table with a good pulse. The inhalation anæsthetic is only necessary when the rib resection proper is commenced, provided morphine and infiltration anæsthetic have been used previously and is given in restricted amounts so that the bronchial and laryngeal reflexes remain in order to facilitate expectoration, while the pain is not felt. An experienced anæsthetist can do with 5–15 gm. chloroform.

A mildly compressing aseptic dressing and bandage is now applied and the patient is put to bed in the prone oblique position. The most important factor during the following hours and days is the behavior of the heart, which, on account of the change in volume of the operated side, has suffered more or less rapidly a marked dislocation in position, and with the collapsed lung it is subjected to the pressure of the air on the operated side. The preservation of the costal cartilages is important in preventing the dislocation from acting too much upon the heart. Figs. 2 and 3 show diagrammatically the diminution in volume of the lungs, due to removal of the bony chest wall. The disturbance in the heart action is similar to that form found associated with severe contusions of the thorax and known as "delirium cordis." It is characterized by increase in the rate, diminution in size, and absence of the pulse. If the heart's action be energetically stimulated with digitalis intraveneously, caffein, and camphor and normal saline solution subcutaneously, the patient is always brought safely through the danger. The course of wound healing is usually smooth. In from 6 to 10 days the drainage tube is removed and healing by primary union is obtained.

Dyspnœa is the rule during the first few days after the operation, due to the fact that the lung on the well side has to do all the work for both, besides being interfered with by the pressure of the air on the opposite lung and the heart. After a few days, however, the breathing becomes normal in frequency. As the wound pain diminishes, expectoration reappears and is facilitated by pressing the hand against the mobilized side, thus aiding the removal of the contents of the caverns.

It is astonishing to note the fall of the temperature and diminution in the daily amount of sputum which follows (see curve). Those that may be interested in the subsequent events that occurred in these cases, I would recommend to read the case histories of my

first three patients presented before the surgical congress and of those treated later, (Bericht der Naturforscher Versammlung in Köln).

In these will be found many details that are important for one to know in order that surprises may not occur during the after treatment. As in Case III the course was complicated on the eleventh day by the rupture of an apical cavern which we feel confident occurred and caused a sudden vomiting of a large amount of tubercular pus. The details of this case will be found in the above. A picture of the way in which the operated side appears will be found in Figs. 5 and 7. The changes which take place in the auscultatory findings in the diseased lung in the following weeks are of great interest from a general pathologic standpoint, as is also the increase in volume which the sound lung undergoes as early as the 10th week after operation. The boundaries of the non-diseased lung on the anterior and posterior aspects of the thorax will be found in Figs. 4 and 8.

It is worth noting that the daily amount of sputum fell from 180–220 ccm. to 25–5 ccm., and that in several cases, months after the operation, it was not possible to find tubercle bacilli. Favorably influenced also were the frequency of coughing and disturbance of the sleep at night, the subjective feeling of comfort and rest, and the body weight, which we found to be increased in all of our cases several weeks before they were discharged.

It is worth noting that the spine was not at all affected during the post operative period; it remained the same as before. No scoliosis develops as after long continued empyemas, with fibrous thickening and contraction of the pleura, because the ribs being removed, no leverage can be exerted on the spine. For some it is necessary that they wear a sort of corset with elastic straps to constantly exert a slight pressure on the operated side.

The end results of our method in ten cases are as follows: Seven of them showed the marked improvements I have previously described, and three died. All of our cases showed extensive unilateral lung tuberculosis in which climatic, dietetic, and all medicinal treatment were of no avail in preventing the spread of the disease; all were feverish and most of them had been confined to bed for months.

The two cases that did not recover were characterized by multiplicity of the tubercular foci, the second one being markedly marantic and suffering with the rapidly spreading form of the disease. The first one of these (Case v) had a cavern in the right lung with tuberculosis of the larynx and left lung; the second (Case vii) bilateral lung tuberculosis with intestinal tuberculosis. The details of these cases with their epicritical remarks will be incorporated in the transactions of the Naturforscher Versammlung in Köln. I mention only the principal points of importance in these cases at present on account of their value in determining the limits of operative interference. In both cases it was very difficult for me to decide whether to undertake such a severe operation or not and I was influenced by the demand of the patients themselves. In the future I would either exclude such cases from an operation altogether or, as I have indicated at the surgical congress, divide the operation into several stages. I might also mention that in the first of these severe cases I only made a partial rib resection and this was followed by severe complications. The expected thick and adherent pleura turned out to be very thin and a small tear occurred in it during the shelling out of the ribs which in spite of immediate tamponade and subsequent purse-string suture resulted in a pneumothorax with concomitant cardiac collapse. The patient lived for several days after but succumbed then on account of difficulty of breathing due to the tubercular larynx, the pneumothorax, and the immediate sequelæ of the operation.

Were I to try to bring together the indications for operation as we have learned them from our experiences so far, we would conclude as follows.

For the operation of total mobilization of the chest wall (thoracoplastic pleuro-pneumolysis) (separation of the lungs from the costal or parietal pleura) those cases are best adapted in which the lung tuberculosis is unilateral, fibro-cavernous in type, occurring in youth and middle age, with variable degrees of fever,

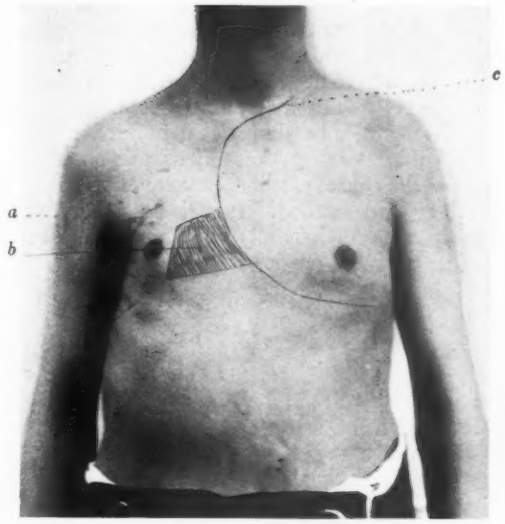


Fig. 4. Case IV. Two and one-half months after operation. *a*, scar; *b*, cardiac dulness; *c*, boundary of lung, well side.

Fig. 5. Case IV. Dashes, spinous processes. Continuous line, percussorial and auscultatory boundaries of the left lung with right-sided operation.

Fig. 6. Case VI. Two and one-half months after operation. Line of suture by incision made only posteriorly. X, apex beat.

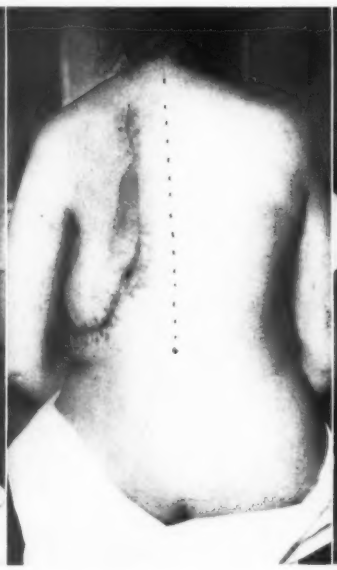


Fig. 7. Case VI. Dotted line, spinous processes.

Fig. 8. Case VI. Two and one-half months after operation. Anterior boundary of the right lung with left-sided operation.

and in which climatic, dietetic, and medicinal treatment are of no avail in staying the progress of the disease. Degree of temperature and amount of sputum have no influence in the placing of the indications. It is very important that the condition of the patient's nutrition be not so reduced that the immediate sequelæ of the operation cannot be overcome. It probably is well to be very conservative in cases where there are multiple tubercular areas, especially when associated with intestinal tuberculosis.

The dangers of the operation lie chiefly in the demands made upon the heart, but in spite of the flabbiness and smallness of the heart of tubercular subjects these difficulties can be overcome by intelligent procedure. When the cases are selected as above the results ought to be almost as definite as in ours if all the operative precautions are observed and in the course of time encourage an extension of the method. It will be some years before we can definitely judge of the healing processes in the lungs.

VISCERAL PROLAPSE

Some New Points in the Etiology

By FRANKLIN H. MARTIN, M. D., Chicago, Illinois
Professor of Gynecology, Post-Graduate Medical School of Chicago; Gynecologist to Woman's, Post-Graduate, and Charity Hospitals

EVER since this subject was drawn to the attention of the profession through Glenard in 1885, much confusion has reigned in the minds of investigators as to the etiology of visceral prolapse, because of the many apparent contradictions presented in the study of that condition.

Recent investigations, which have been forced upon the clinician because of the growing recognition of the importance of the subject, have multiplied facts from observation and experience upon which we can now begin to base a rational explanation of the apparent incongruities.

A careful consideration of the accumulated data, I believe, enables us to declare that the tendency to general visceral prolapse of the abdomen, the pelvis, and the chest occurs under two markedly different defects; viz., a defect or retardation of the evolutionary development of the body or from congenital developmental defects; and second, from the effects of acute or chronic changes, including traumatisms, upon the normally developed body. Hence, we may affirm that the tendency to visceral prolapse is due (1) to developmental defects and (2) to acquired defects. In this communication I proposed to discuss duly the first division of the subject; viz., developmental defects.

Developmental. Evidence of an incontrovertible character has accumulated which demonstrates that man at his highest point of development in the evolution of the animal kingdom, in perfect health, is free from visceral prolapse or displacements. As he recedes from this perfection of development and exhibits reversions to or inheritance from the stages below in the degrees of animal development, he begins to exhibit signs of visceral disturbances.

In order to obtain convincing evidence that certain developmental defects show a tendency to certain visceral changes, it is only necessary, I believe, to investigate a series of cases from the highest state of development down through the various developmental defects shown in the human animal and to compare the defects found with the visceral condition exhibited.

Normal Development. The highest type of the normally developed human form, it probably would be conceded, would be a composite of the men and women of perfect health between the ages of twenty and thirty years, of that state of society the progenitors of which had been engaged in the normal pursuits of life.

Fig. 1.

Fig. 2.

Fig. 1. Outline sketch of approximately a correct figure of a normally developed female.

Fig. 2. Outline sketch of approximately a correct figure of a normally developed male.

Fortunately we are able to get practically this in graphic outlines constructed from measurements obtained in the anthropometrical records of the large colleges, as Vassar and Smith for women and Harvard and Yale universities for men. (Figs. 1 and 2.)

By comparing these with the ideal as represented by the artist in the Venus de Milo, (Fig. 3) and Michael Angelo's Dorid (Fig. 4), we find a substantial agreement between them, showing that the human form had reached its full development in those early days and that the artists of those times recognized and accurately depicted the normal.

The skeletons (outlined in Figs. 1 and 2) are copies representing as accurately as could be obtained by comparisons and measurements, skeletons which would represent the bony frames of the composite bodies of 1 and 2 respectively.

Lower Forms of Development. In making comparisons between the human form and the lower animal forms, I will for the present convenience compare the skeletons showing the body and the arrangement of the viscera and peritoneum; and also will confine my comparisons only to a few of the animals next below the human in the line of ascent, and the developing human as shown in the fœtus and the child, as only these furnish us lines of similarity with the defectively developed human adult.

I

BODY OUTLINE AS SHOWN IN SKELETON

Fig. 1 represents the skeleton of a normally, well developed adult female; Fig. 5 that of a typically defective development of a female who was the possessor of general visceral prolapse; Fig. 6 represents the skeleton of an adult male gorilla; Fig. 7 represents the skeleton of a male ourang-outang; Fig. 8 that of a monkey; Fig. 9 the skeleton of an embryo at four months.

By a cursory comparison of the drawings shown a distinct discrepancy between them is easily recognized. Figs. 1, 2, etc., show drawings of the normal human in which the viscera are maintained in their normal positions and relations while the animal maintains throughout the activities of adult life the perpendicular position. This cursory examination shows conspicuously (*a*) a marked tilting backward of the sacrum, changing the general axis of the sacrum from a line parallel to the axis of the body to a line at nearly right angles to the body, with its anterior inner surface facing downward and forward, and the posterior surface facing upward and backward. The angle between the plane of the inlet of the true pelvis of the normal (Fig. 10) is 55 to 60 degrees with the horizontal, and the angle of the plane of the outlet is about 10 degrees. Both of these planes look upward and forward. A line projected forward and upward at right angles to the center of the plane of the inlet of the normal pelvis would strike the umbilicus and the lower projection will touch the middle of the coccyx.

If this is compared with Fig. 5 representing the undeveloped female and Figs. 6 and 7 representing the higher lower animals, and Fig. 9 representing the embryo, the pelvis will be seen to show a decided variation with a gen-

Fig. 3. Profile view of Venus de Milo.

Fig. 4. Profile view of Michael Angelo's "David."

eral tendency to become perpendicular with the axis of the body, with the subject in an upright position.

The normal human skeleton shows on examination, of the anterior line of the bodies of the vertebræ of the spinal column, a quite marked curve forward beginning with about the tenth dorsal vertebra and extending to the first sacral and reaching its maximum at about the third lumbar.

This front convexity, taken in connection with the front concavity and backward extension of the lower end of the sacrum and the front concavity of the dorsal spine extending from the tenth dorsal upward to the seventh cervical, gives rise to a marked hollowing of the back in the lumbar region, a curve backward of the upper portion of the trunk and shoulders, and again a forward curve or concavity at the back of the neck. On the other hand in the illustration representing the non-developed woman, the higher monkeys, and the human embryo and child, the forward curve of the lumbar region is much less or absent. The highest developed lower animal, the gorilla (Fig. 6), shows a slight tendency to this curve. There are other points of difference which are interesting but not so relative to our subject, which may be observed, such as the difference in the length of the arms, the difference in the length of the pelvis, the location of the acetabulum, the frame of the chest, the contour of the abdomen, the forward bend of the body and the difference in the axis of the body, and a change in the center of gravity.

The body form as shown in comparison be-

Fig. 5. Sketch of non-developed female, with marked enteroptosis. Note straight back, low projecting "cannon ball" abdomen and perpendicular pelvis.

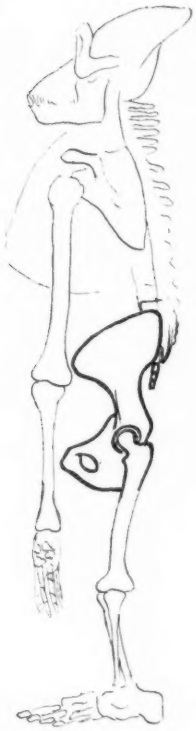


Fig. 6.

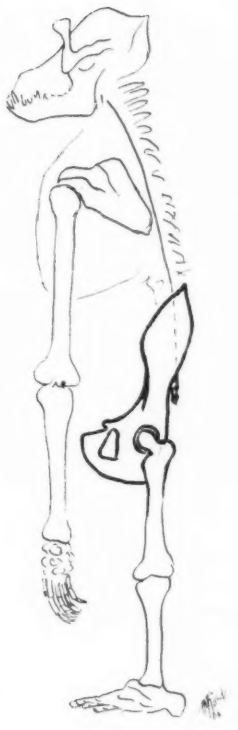


Fig. 7.

Fig. 6. Skeleton of an adult male gorilla. Note slight curve in back and perpendicular sacrum.

Fig. 7. Skeleton of an ourang outang; lumbar curve entirely absent.

tween the normal adult (Fig. 1) and the defective adult (Fig. 5) shows a narrowing of the chest in the latter with a downward tilt of the ribs carrying the sternum downward and backward nearer the spinal column and thus lessening the capacity of the chest. The waist is small and the upper abdomen flat while the lower abdomen is protruding and pendulous. The individual has a forward bend of the body, the arms and shoulders project forward, while the forearms and hands swing forward of the line of the axis of the body.

To show the relation of these differences of form to our subject, we must point out the fact that Fig. 5, representing the human female, exhibiting changes of form varying from the normal human and approximating the form of the lower animals, is a type which in the human almost invariably indicates a tendency to visceral prolapse. This is proven by obtaining the index as advised by Beecher and Lenhoff, and Harris, and by direct observations in the examination of cases.

On the other hand the animals possessing the peculiarity of form exhibited by the defective human subject, we must take for granted have normal visceral support. Their body forms represent the highest development of their type, exactly as Fig. 1 represents the highest development of the human form.

While there are many factors entering into the subject, probably one of the most important reasons why the viscera behave differently in the lower animals under consideration, than in the human whose defective development makes her resemble the lower animal, is because the lower animal does not actually maintain the perpendicular position. The most

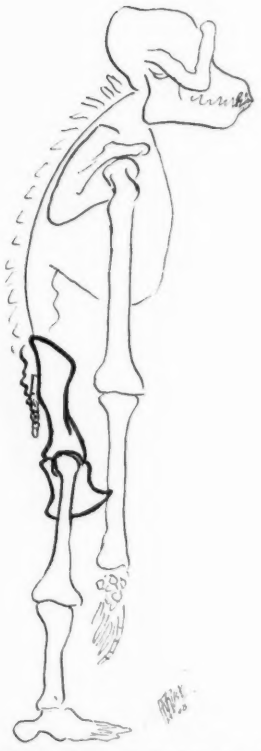


Fig. 8. Skeleton of one of the lower monkeys. Note convexity instead of concavity of lumbar region of spine.

man-like ape in almost all his movements assumes a forward inclination of the body, and while he does not assume the actual position of a quadruped, he employs his upper long extremities for grasping surrounding objects in order to maintain his balance and to aid in locomotion. While resting it is always with the thighs flexed on the abdomen and an acute forward bending of the body (Fig. 11) and as he departs more from the human as shown in my illustrated cases, he assumes more and more a horizontal position. Hence the supports of the viscera of the lower animals are differently arranged and comport differently with the change in the body position. The defective human, on the other hand, is a perpendicular animal and must maintain the upright position, and besides is defective in the supports, as for example the strong fascia lata of the abdomen afforded to a normal animal of her apparent type.

II

THE VISCERA AND PERITONEUM AND OTHER SUPPORTS

Before considering further the bearing of developmental defects on the production of visceral prolapse, it will be necessary to review the subject of visceral support as maintained in the normal individual and account for its perfection in the healthy man.

The hollow trunk containing the viscera is a semi-flexible closed cavity the shape of a hollow, flattened cylinder, with rounded ends. Its upper third is a casement protected by bony ribs, interwoven with muscles with the power to expand or contract; its lower end is an immovable casement of bone, while its central major portion is large and constructed of muscles and elastic tissue reinforced by the sustaining spinal column of bone, and this trunk has for its principal object the maintaining of a comfortable snugness between its walls and its contents or viscera, which in the performance of their several functions constantly vary in capacity and position (Fig. 12).

Fig. 9. Outline of body and skeleton of human embryo.

Visceral Supports. In examining the viscera in reference to their support, we are struck by the magnificent adequacy of the devices furnished for their maintenance in a range of approximately normal locations which, at the same time, allow of untrammeled freedom of action in the performance of their complicated functions.

Without taking into consideration a highly developed sympathetic nervous system which automatically and instantly takes cognizance of, and adjusts all antagonism of functional activity between the viscera, it would be impossible to account for the harmony existing between them.

Visceral Shelves. Observing the viscera as a whole in their relation to the trunk, within which they are maintained, we are struck by a series of strong shelves, which definitely and more or less immovably sustain the principal ones in their several positions. These shelves are more properly the broad surfaces of attachments which make shelves of their attached viscera, rather than actual projecting ledges, as a shelf would properly indicate. They are the areas between the viscera and the parieties where the peritoneum has blended in the process of development and become obliterated and been displaced by strong connective tissue unions between the opposing surfaces.

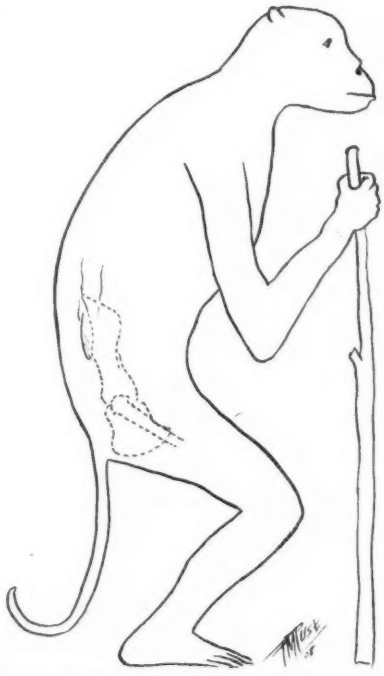


Fig. 11. Outline of higher man-like ape, showing normal attitude.

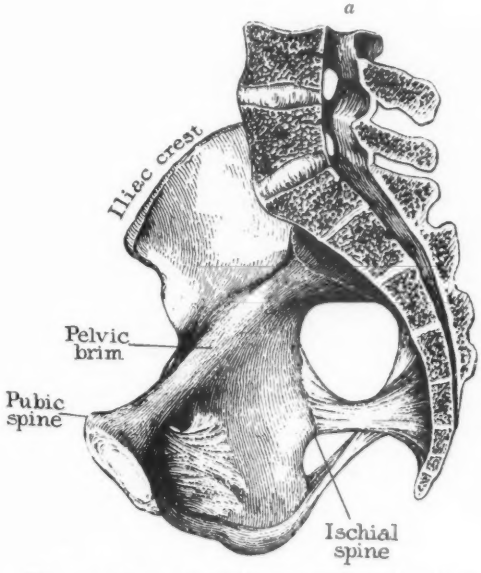


Fig. 10. Normal inclination of female pelvis. (After Eden.)

As we observe these shelves in almost continuous series of lines from the diaphragm to the pelvis (Fig. 13), we may rapidly enumerate them as (*a*) the large triangular space on the superior and posterior surface of the liver, (*b*) the small triangular uncovered space on the stomach near the œsophagus; (*c*) the second portion of the duodenum with its firm attachment sub-peritoneally from the second to fourth lumbar vertebræ; (*d*) the broad attachment sub-peritoneally of the pancreas; (*e*) a triangular connective tissue area connecting the top of the spleen with the diaphragm; (*f*) the connective tissue union between the ascending colon and the parieties at the point of obliteration of its meso-colon; (*g*) the similar attachment of the descending colon at the point of elimination of its meso-

Fig. 12. Drawing from Schultze & Stewart, showing trunk with molded organs in situ.

colon; (*h*) the general forward projection of the spine near the lower end of the middle zone; (*i*) the flaring upward faced concave iliac bones, and finally (*j*) the pubis with its strong attachments of ascending pyramid of muscles and retaining fossæ.

Retaining Fossæ. Upon observing carefully the arrangement of the abdominal viscera especially in reference to their supports, we are struck by the fact of two or more strong retaining concavities facing upward which serve to shelve up the organs lodged in part or wholly in them. (*a*) The most important of these is the space at the sides of the spinal column extending from the ninth or tenth rib to the second lumbar vertebra in front of the psoas and quadratus lumbar muscles between and behind the peritoneum, the space hollowed out for the capsules of the kidneys. With the normal forward projection of the lumbar vertebra, the lower portion of this fossa with the individual in the perpendicular, projects forward and the concavity looks upward as well as forward. (*b*) Next in importance to the kidney fossa are the fossæ already referred to, the flaring concavity presented at the lower end of the lower zone of the trunk; viz., the iliac fossæ. There are several other retaining fossæ less evident, as the splenic fossa, and that moulded too by the second portion of the duodenum and the pancreas. This latter attachment is one of the strongest and from its central position and its sub-peritoneal location is the most invulnerable bulwark of the visceral supports. (Fig. 14.) The strong sub-peritoneal attachment of the duodenum, the whole length attachment of the pancreas, arising from their deep vessel attachments, the cœlic axis vessels interwoven between the two, all covered and protected with the tent-like peritoneum deflecting from the mesentery of the transverse colon, make a strong hold that has been well named the "axis mesenterica."

By observing the skeleton from behind (Fig. 16) it will be seen that the inward curve of the lower ribs furnishes when in their normal position, a retaining fossa for the liver, the spleen, and the upper portion of the kidneys.

Supporting Ligaments. The supporting ligaments of the liver are peritoneal deflections which, with the large triangular uncovered

portion of the organ lying in contact with the diaphragm in the normal state, cause the liver to snugly hug the upper end and posterior portions of the vault of the abdomen. Besides the peritoneal deflection underneath the peritoneal investment of the liver is a fibro-elastic membrane which is intimately adherent to the peritoneum and is continuous over the liver where the peritoneum is deficient. At the portal fissure this fibrous layer invests the hepatic vessels, forming a special sheath which follows the vessels as they enter the liver. (Morris.) Everywhere the peritoneum deflections are reinforced with this strong fibro-elastic tissue.

The stomach is snug between its fixed cardiac end and its pyloric end by the gastro-hepatic ligament which holds it beneath the liver and through the liver attachment to the diaphragm. To the left of the cardiac attachment on the greater curvature, its peritoneal deflection attaches it decidedly to the diaphragm (gastrophrenic ligament) and further

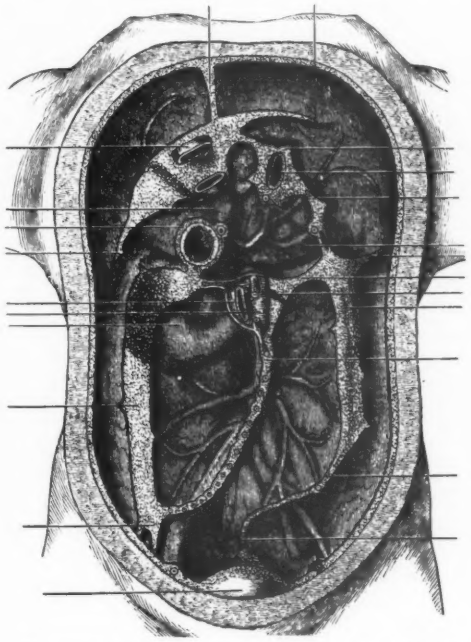


Fig. 13. (Morris.) Showing extent of obliteration and blending of peritoneum, giving firm attachment to the different viscera to the posterior parieties, in the human adult.

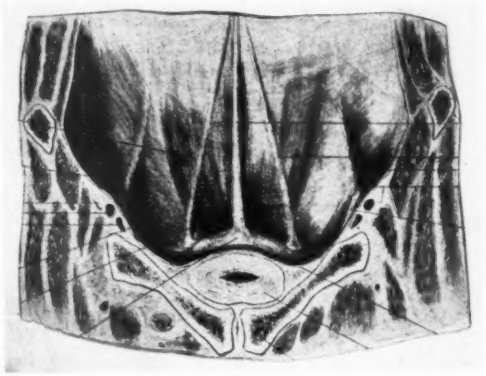


Fig. 14. (Piersol.) Showing fossæ on the peritoneal surface of the anterior abdominal wall immediately above the pubis.

to the left onto the spleen (gastrosplenic ligament). The peritoneum from the greater curvature descends in two layers to form the great omentum, returns and after fœtal life in the normally developed, attaches to the transverse colon and then blends with the superior surface of the transverse meso-colon and becomes continuous with the parietal peritoneum of the lesser cavity.

Mesenteries. The mesenteries are supporting structures of great value and have an unusual bearing on the subject of visceral prolapse. The development of the mesentery in embryo should be studied. At about the fifth month of fœtal life, an obliteration of the mesentery between the ascending and descending colon occurs by a blending of the peritoneal surface of the mesocolon with the parietal peritoneum and finally with a displacement of the peritoneum by a broad surface of fibro-elastic tissue which fixes these two respective portions of the colon to the posterior abdominal parieties. In about one-fourth of the subjects, this blending fails to occur and these portions remain with mesenteries of varying lengths. This fixation of the colon when normal, extends to the hepatic flexion on the right and begins at the splenic flexion on the left, giving substantial support to the two ends of the transverse colon between which it swings and maintains the transverse meso-colon as with its blending with the posterior layer of the great omentum, it affords a hori-

Fig. 15. (Schultze & Stewart.) Showing arrangement of the organs in normal position, particularly the high position of the transverse colon and its diaphragm-like support of the superficial organs.

zontal ledge or partition separating the greater and lesser peritoneal cavity.

When normally developed the transverse colon with its mesentery, with its high fixed flexions at either end (hepatic and splenic) affords a strong diaphragm of support to all the superimposed organs. (Fig. 15.)

The mesentery of the small intestines in its attachment extends in almost a straight line from the left of the front of the second lumbar vertebra to the right sacro-iliac joint, a distance of about six inches. The length of the mesentery from its attachment to the abdominal parieties to its attachment to the intestines or between its two borders is four to six inches at its beginning, eight to nine inches at the middle of the intestine and one, two, or three inches at the lowest portion of the ileum. While the attachment of the mesentery to the parieties is only six inches in length the outer border or the intestinal attachment is twenty feet or more. Of necessity this rapid expansion from a fixed attachment of one-half a foot to an attachment of twenty feet, at its intestinal border, in an average length of not more than eight inches, shows a marvelous capacity of folding and as these expanding folds are crowded together one can readily understand their definite supporting power as they joist-like brace the intestines against the opposing abdominal walls.

Ligament Supports of the Pelvic Viscera. The floor of the male pelvis is so strongly fortified with muscular and especially strong fascial supports which are never called upon to yield in the process of parturition and which are not weakened by the parturient canal, that it is seldom the seat of troublesome prolapse.

In the female pelvis the bladder, the uterus, the vagina, and the rectum are all subject to troublesome descent. The female bladder has a broad sub-peritoneal attachment or shelf upon which it may rely for support if the uterus and bladder, with which it is closely associated, are normally supported.

The uterus is supported laterally with two mesenteric-like folds, between which it swings, its broad ligaments. Its neck or cervix is maintained normally well back close to the hollow of the sacrum by the normal pelvic fascia and the sacro-uterine folds of peritoneum and their connective tissue, while the round ligaments act as forward guy ropes in the attempted excursions of the fundus backward. The floor of the pelvis has the same strong fascial supports as those of the male, but is at a disadvantage in supports proportionate to its greater area, and also in sustaining the additional opening, the vagina.

Muscular Trunk. Outside the peritoneum the sub-peritoneal elastic connective tissue, the sub-peritoneal layer of adipose tissue, we have a casement of muscles bound together with aponeurosis and strong fascial sheaths and external to this (with the exception of the diaphragm) the protecting integument, the sub-integumentary fat and other layers of elastic supporting tissue. This muscular casement is extremely movable in the diaphragm

and the front abdominal wall and these two co-ordinate in maintaining a close relation between the visceral contents and the cavity of the trunk. If the diaphragm is depressed there is a corresponding expansion of the other movable muscles of the abdominal wall. The only exception to this is when the elastic hollow viscera themselves vary in their contour from being distended or collapsed.

Determining Fossæ. The peritoneal surface of the movable anterior abdominal wall is divided into a number of shallow but well margined fossæ, representing the spaces between aponeurotic lines and the varying thickness of the different portions of the muscles. (Fig. 16.) These fossæ act as vantage points as the viscera are manipulated and maintained in their relative positions by the ever acting abdominal muscles which are ever on the qui vive when under the influence of a normal state of sympathetic nerve control, to come to the relief of refractory viscera and restore their normal relationship. These fossæ act as intelligent mortises on the wall of the structure to support the ends of the joist-like mesenteries with their attached intestine, the other ends of which are hinge-like attachments to the posterior abdominal parieties, and thus assist in establishing a series of stories and floors in the abdominal architecture.

Fat. With fat, we must deal in considering the supports of the abdominal viscera. Fat as a sub-peritoneal layer aids in padding the parieties and maintaining a necessary relative snugness beween the reservoir and the contents. Fat in the various peritoneal ligaments separates their layers, stiffens their consistency, pads the blood vessels which traverse them to their respective organs. Fat in the mesenteries acts similarly. It stiffens them, renders them more supportive, renders them less flexible, shortens them by separating their layers and protects their multitude of blood vessels by its padding effect.

All sub-peritoneal organs and all organs partially sub-peritoneal, or viscera, have their point of peritoneal deflection filled with fat. The kidney without its normal fat is like a pea in a pod, loose and movable, and entirely dependent upon the elasticity of its fascial and muscular layers about it which, if together with the connective tissue surrounding it, are not unusually responsive will leave the organ unsupported and destined to prolapse. About the duodenum, the pancreas, the spleen, the liver, the ascending and descending colon attachments, the bladder, this cement of fat is filled to stay, pad, stiffen, insulate and enormously support.

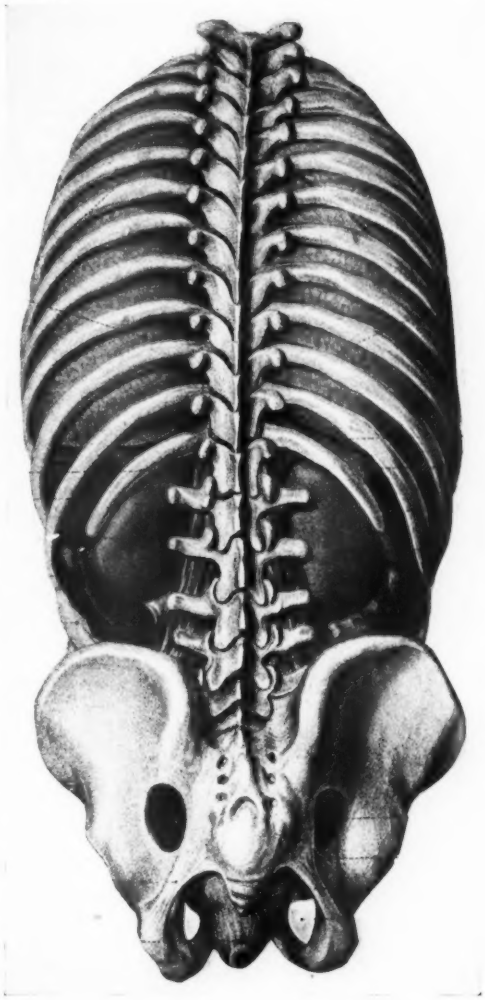


Fig. 16. (Schultze & Stewart.) Note fossæ made by in-curving lower ribs, supporting the kidney, liver, and spleen.

In studying the lower vertebrates from the

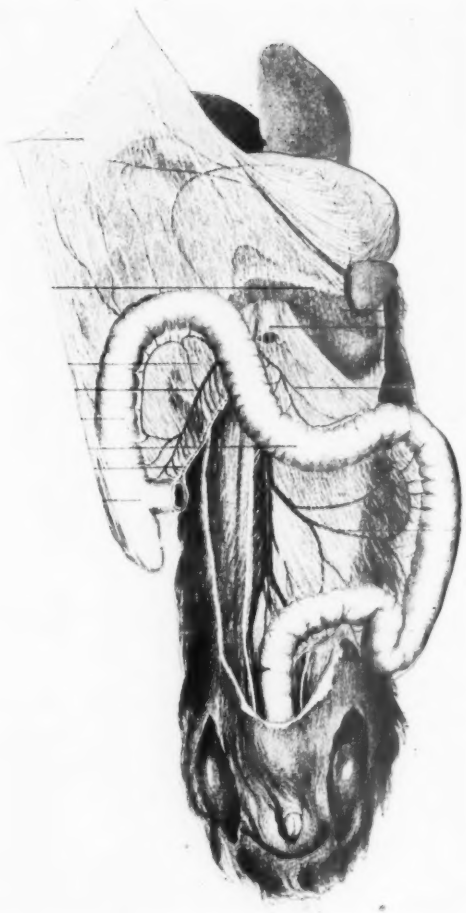


Fig. 17. (From Huntington.) Showing disposition of mesentery only partially rotated, long mesentery of ascending and descending colon, failure of firm fixation at hepatic and splenic flexure. (Rhesus monkey.)

higher monkeys down and comparing the disposition of the peritoneum in these animals as in the process of evolution it develops to perfection in each animal, with the perfected peritoneum in the normal adult human, there will be found a distinct difference in its final behavior due to the wide difference in the supports required for the abdominal viscera in the two classes of animals, the one a quadruped with a horizontal body, the other a biped with a perpendicular body.

Development of the Peritoneal Support. In studying, too, the stages of development of the peritoneum in the human embryo to its final perfected state in the normal human adult, it will be found that its different stages can be found duplicated as the final normal condition of some one of the many lower animals.

For that reason it is obviously impossible to say when a defect in development of the peritoneum and the disposition of the abdominal organs is discovered whether it is an arrest of the development from embryo or a reversion to a primitive type in the process of evolution.

For comparison Fig. 17 from Huntington, representing the peritoneal disposition in relation to the viscera may be taken as an example of its disposition in the quadruped or lower animal. Fig. 18 shows the peritoneal disposition in the human. The changes noted in the closing of this gap in evolution may be enumerated briefly as follows:

(*a*) Complete rotation of the large gut with a distinct mesentery in the human; failure of

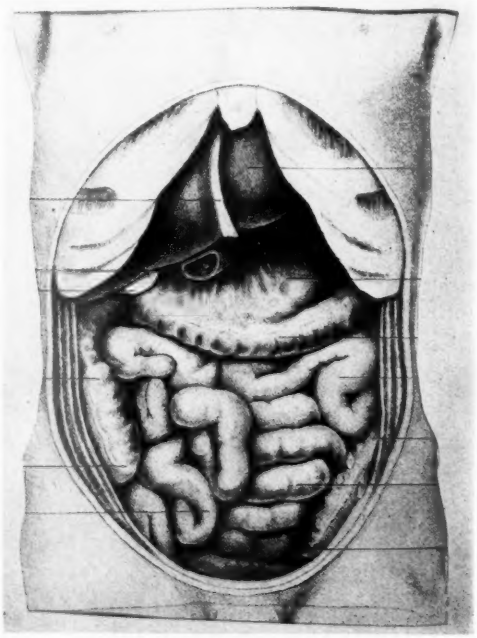


Fig. 18. (From Piersol.) Showing disposition of large intestine in adult human.

the gut to rotate and a mesentery in common with the small intestines in the lower animals.

(*b*) Obliteration of the mesentery of the ascending colon by blending of the original meso-colon with the parieties and final obliteration of its layers in the human; failure of the meso-colon to blend and to become obliterated in the animal.

(*c*) Obliteration of the mesentery of the descending colon by blending of the original meso-colon with the parieties and final obliteration of its layers in the human; failure of the meso-colon to blend and to become obliterated in the animal.

(*d*) Firm fixation of the hepatic flexure and the splenic flexure of the colon in the human; failure of fixture of hepatic and splenic flexure to become firmly fixed in the animal.

(*e*) Immovable fixation of the duodenum and pancreas to the posterior parieties by blending and obliteration of its mesentery and parietal fixation in the human; failure of the duodenum and pancreas to become fixed (even retaining a considerable mesentery in cases) in the lower animals.

(*f*) Firm fixation of the liver by large triangular area to diaphragm and parieties by blending and obliteration of its early peritoneal covering over considerable space, making it a partial sub-peritoneal organ; failure of such obliteration of peritoneum in the lower animals leaving the liver with long peritoneal attachments.

(*g*) Fixation of triangular space at cardiac end of stomach by blending and obliteration of peritoneum; fixation of spleen by blending and obliteration of original peritoneum; shortening of the mesentery of the small as well as large intestine by folding, blending, and obliteration of a portion of its parietal attachments; a firm sub-peritoneal fixation of the general parietal peritoneum by a close fibro-connective tissue attachment in the human; failure of such development in the lower animal.

III

SOME COMMON DEVELOPMENTAL DEFECTS

Skeleton. I have endeavored to depict the normal fully developed outline of the body as shown in the skeleton. (Fig. 1.) The soft parts built around such a skeleton usually conform to a condition of normal arrangement of the viscera when the individual is in health or has not been subject to traumatism. This has been demonstrated and confirmed by observation and a standard of measurements have been devised which aids us in reliably arriving at our conclusions.

To recapitulate, the principal points which enable us to at once recognize the defective skeleton are, first, the straight spinal column, the flattened chest walls with slanting ribs and the perpendicular pelvis. (Fig. 3.) The individual built around such a skeleton has a straight back, a small waist in comparison with the pubic and clavicular line and chest measurement, a broad protruding lower abdomen, a slightly forward bend of the neck, long arms swinging forward of the hips and the center of gravity of the body in a line in front of the pubis. This individual possesses soft flabby muscles, the outline of the body lacks symmetry, the fossæ are weak, and the adipose tissue is soft and deficient

It is my belief based on numerous observations in abdominal operations on patients of this type that these individuals possess defects in the final development, in detail, necessary to its highest degree of efficiency in the human, of not only the skeleton and outward body but also of the peritoneum and its accessories, as the mesenteries, visceral ligaments, and parietal blendings.

We have observed that the highest developed peritoneum of the human passes through all the successive stages of development in its transition from the embryo to its perfection in the adult, that are found normally in all lower vertebræ, and then still further to a contion of perfection necessary to meet the exigencies of the human biped.

If, in the process of development of the peritoneum in the human, an arrest occurs, at any point of the progress before the normal development has been reached, a defect of support protection and possibly function will likely occur in the viscera affected by such arrest.

The conspicuous defects that I have noticed are: Failure of blending and obliteration of the peritoneum between the liver and the parieties. Failure of obliteration of the mes-

entery of the ascending colon. Failure of obliteration of the mesentery of the descending colon. Failure of peritoneal blending at the hepatic flexure of the colon. Occasional failure of peritoneal blending at the splenic flexure of the colon. Failure or partial failure of the large intestine to rotate, leaving it with a primitive meso-colon. Failure of blending of the peritoneum of spleen to parieties. Failure of posterior layer of omentum to blend with transverse colon. Occasionally, but rarely, remains of a duodenal mesentery and failure of that gut to become fixed. Failure of the pancreas to become fixed by a persistency in its parietal peritoneum. Failure of the parietal peritoneum everywhere to become normally fixed to the parieties, a preternatural loosening of attachment being apparent. Failure of the mesentery of the small intestine to spread out, blend, and become partially obliterated, and hence it remains preternaturally long.

The type of individual I have endeavored to describe exhibits frequently an abnormal condition of the nerve supply to the trunk. The motor and sensory nerves of the cerebrospinal system respond slowly or erratically to stimulation. The automatic sympathetic system, including the vasomotor control respond slowly to stimulation and are slow to react from such attacks. The rhythm of perfect attunement and adjustment between the viscera and their supports and the contained parieties, which should exist with the highest state of development in health, is therefore lacking.

This muscular insufficiency is one of the marked characteristics that we immediately observe in these individuals. The anterior abdominal wall hangs pendulent, its muscles failing to respond to their function of keeping the contents of its cavity snugly nursed together, and at their proper level. A reversion to the hammock-like state of the strong fascia supported abdomen of the quadruped where a continual dead weight of otherwise unsupported organs is to be carried instead of the highly developed muscular walls of the biped, which are called upon to do an intricate, intermittent work under the stimulus of an acutely developed nervous mechanism in maintaining organs otherwise partially supported in their normal relations. Or they hang pendulent and loose because of their inability, unsupported by the strong lateral fascia of the quadruped, to maintain the dead weight of the peritoneal organs of a biped which have failed to develop their normal supports. Or both of these defective conditions may, and doubtless do, frequently exist in the same individual suffering with abdominal visceral prolapse.

The above defects are so frequently found in the type of body that I have described as indicating a lack of development and also in connection with visceral prolapse that I have learned to look for them together. I am aware that it will take a long series of carefully recorded cases to establish definitely my contention that there is a strong etiological factor in the conditions I have endeavored to describe, and general visceral prolapse.

CONCLUSIONS

1. Defects in the growth of the human body arising as a result of an arrest at some point in the development from embryo (congenital) or as a result of reversion to a lower type (evolutional) are strong factors in the etiology of visceral prolapse.

2. The most conspicuous defects are observed in: (*a*) development of the skeleton (*b*) the development of the peritoneum; (*c*) the development of the muscles of the trunk; (*d*) the development of the fascial layers of the trunk; (*e*) the development of the efficiency of the nervous apparatus controlling the viscera and their relation to the trunk; (*f*) the development of fat layers and deposits.

3. The conspicuous signs of these defects are exhibited in: (*a*) a straight spinal column showing little or no forward curve in the lumbar region, a perpendicular sacrum, a forward bend in the cervical region; (*b*) a flat chest, a small waist, perpendicular pelvis, protruding of the lower zone of the abdomen; (*c*) slightly forward bend of the trunk, stoop shoulders, long arms.

NOTE.—Two interesting articles bearing out my contentions have been written by Dr. Eliza Mosher in the New York Journal of Gynecology and Obstetrics, November, 1893, and Dr. Agnes Vietor in the Boston Medical and Surgical Journal, August 2, 1906.

THE COMPARISON OF THE TOTAL UREA EXCRETED BY EACH KIDNEY IN SURGICAL DISEASES OF THESE ORGANS[1]

Its Value in Estimating the Functional Capacity and Pathological Changes of Kidneys

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THE importance of an accurate estimate of the kidney function in surgical diseases of these organs has probably furnished the incentive for as great an amount of careful work as any other modern surgical problem. The problem to be solved is so complex a one that we could hardly expect any one test to be valid in all cases. Thus, while many methods have been exploited which are of much value, no one seems to be adequate in every instance.

With the advance of kidney surgery many cases considered inoperable ten years ago are operated upon to-day. With this advance, the tests of functional kidney capacity must be constantly changing. The borderland class contains fewer cases.

By the normal functional capacity of a kidney we mean primarily the ability of that kidney to assume the total excretory function after extirpation of its fellow kidney. In other words, the functional capacity is a measure of the "*reserve force*" of a kidney.

A number of observers have lately sought to determine the effect of disease of one kidney upon the working power of the other. Beer (1) has found in several instances. for example, that the presence of the diseased kidney caused a much retarded excretion of phloridzin, which excretion returned to normal after nephrectomy of the diseased kidney. It would seem inaccurate to say that the sound kidney was doing less work before the nephrectomy than after. In reality it probably was doing more work before the removal of the diseased kidney. But its reserve force was slowly decreasing. It was being brought nearer to the point where nephrectomy of its fellow kidney would be dangerous, if not fatal. Therefore, I think we are right in saying that the functional capacity is a measure of reserve force, and not of total work. The reserve force, I believe, varies inversely with the total work done by a kidney.

The method I wish to present is the comparison of the total urea excreted by each kidney during the same period of time. In other words, the percentage of urea multiplied by the quantity of urine excreted.

To-day the large majority of the methods used to prove the functional capacity of the kidneys depends upon the introduction of foreign substances (phloridzin, methylene blue, etc.) into the circulation and the observance of their excretion. Often simply the time of excretion is observed. If within a certain time after the injection of phloridzin into the body, sugar appears in the urine a kidney is said to be functionating normally. Often the estimate of the functional capacity depends upon comparison of the amount of coloring matter — as, e. g., indigocarmine — excreted by each kidney.

The phloridzin method, perhaps the most popular of all, has been proven to mean nothing when negative. Alessandri (2) of Rome concludes, "The phloridzin test is good if in harmony with other tests; alone it is of little value, especially if negative." Beer of New York has shown that while the phloridzin test is of value when positive it is of no value when negative.

The determination of kidney sufficiency or insufficiency by the examination of the kidney secretion, the urine, and particularly by the nitrogenous compounds in the urine, has fallen into disrepute. Caspar says, "A determination of the urea excreted, or more correctly, the total amount of nitrogen without controlling the amount of N. in the diet, or consideration of the N. excreted by the intestines, does not allow one to draw the slightest conclusions," and "even the results

[1] Read in part before the American Urological Association, at Chicago, June 1, 1908.

obtained by exact means give no reliable information as to whether the function of the kidneys is sufficient or not." Von Noorden, (4), however, makes the following statements, ... the elimination of certain drugs (iodid of potassium and methylene blue in particular) frequently proceed in an altogether different way than the elimination of normal metabolic products." "From all the results that have been published we learn that the determination of the excretion of certain chemicals that can be readily recognized in the urine may occasionally be of value in interpreting the significance of drug treatment in diseases of the kidneys, and may also possibly form the basis of certain theoretical conclusions; but, at the same time, we find that these studies do not by any means permit us to draw any definite conclusions in regard to the power of the kidneys to eliminate normal excretory products. The only way in which to determine the power of the kidneys in the latter respect is to adopt the much more tedious method of testing the elimination of these products themselves."

The reason for the failure of urea elimination as a functional kidney test is, perhaps, that it has been sought to determine the absolute index of the functional capacity of one kidney without considering the work done by the other kidney. And also that it has been the *percentage urea rather than the total urea* that has been largely dealt with.

Albarran (3) has done a large amount of work in this subject, and while he believes that the comparison of the total urea, $\Delta \vee$ or total sugar is much better than the % urea, Δ or % sugar, in estimating the functional capacity of the kidneys, he believes that error may arise when any one of these is used; and that his "experimental polyuria" is the only accurate way of estimating the kidney function.

In this paper I shall attempt to prove my deductions entirely by a comparison of the total urea excreted by each kidney during the same period of time. I wish to emphasize this comparison of the total quantities of any substances excreted by the kidneys in estimating the functional capacity. For instance, in the phloridzin test, instead of simply forming conclusions by the fact that the urine shows a sugar reaction in so many minutes, the total quantities of sugar excreted by each kidney should be determined and compared. This comparison necessitates the accurate collection of all of the urine from each kidney, during the same period of time, and as far as phloridzin is concerned, over a longer period of time than the thirty or forty minutes usually consumed in this test.

I shall not draw any conclusions, except in a few normal cases, as to the value of this total urea method by comparing it with other methods — phloridzin, methylene blue, etc., — nor shall I consider the normal excretion of phloridzin, etc., as a criterion of normal kidney function. In the cases in which the total urea method has been used, *the sole proof of renal sufficiency or insufficiency will be nephrectomy of one kidney, with recovery or death from renal insufficiency following, thus proving the functional capacity or lack of capacity of the remaining kidney.* This is, of necessity, the only way which the value of any test of kidney function can be determined.

My conclusions are based upon the critical examination of 78 cases gathered from the literature and my own work, in 77 of which nephrectomy was performed, and in one nephrotomy. It might be mentioned that in the important work of Caspar and Richter, "Functional Diagnosis of Kidney Disease" (1903), which has done much to establish the value of the "phloridzin method" in functional kidney diagnosis, *in but 7 of the 90 cases reviewed was nephrectomy performed.*

Of the 78 cases I have reviewed, 74 recovered, 2 died of renal insufficiency, and 2 from other causes following the operation. Because of the comparatively small number of cases reviewed any conclusions that may be drawn will be tentative, and further work in this line will be needed to verify these conclusions.

What I shall attempt to establish is:

1. To determine in what cases nephrectomy may be performed without fear of death from renal insufficiency ensuing.

2. To indicate a borderland class of cases where the ability of the comparatively sound kidney to functionate after nephrectomy of its fellow is questionable.

The subject will be considered under three different headings:

1. Methods for obtaining the urine from either kidney.

2. The value of the comparison of the total urea excreted by each kidney in estimating the functional capacity of the kidneys.

3. The value of this comparison as indicating the pathological changes in the kidneys.

1. METHODS FOR OBTAINING THE URINE FROM EITHER KIDNEY

In estimating the total urea excreted by each kidney, it is of course most important to know that we are accurately collecting the *total quantity* of urine excreted by each kidney during a given period of time.

One of the following methods has been used to collect the urine from either kidney:

1. Ureteral catheterism; or

2. Collection of the urine by the Luys' Separator.

It seems hardly necessary to mention that the collection of the urine from the kidneys, by whatever means, *must be simultaneous.*

The errors which may arise when either the ureteral catheter or Luys' Separator is used readily divide themselves into three classes:

1. Mechanical: Those arising from a faulty instrumentation, and resulting in either failure to collect all of the urine flowing from each kidney; or causing a mixture of the urines.

2. Reflex oliguria or polyuria, caused by the instrumentation.

3. Collecting the urines at inappropriate times.

1. Mechanical errors. We have not time to discuss this question at length. With care both ureteral catheterism and the Luys' Separator give accurate results, and all of the urine may be collected from either side.

Probably more care is necessary with the ureteral catheter than with the Luys' Separator, as with the former, in a certain number of cases, an extra-catheter flow occurs. In the 78 cases ureteral catheterism was used in 38 and the Luys' Separator in 40. Those in which the Luys' Separator was used gave, I believe, absolutely accurate quantitative results.

Those in which the ureteral catheter was used are somewhat open to criticism. I believe two factors are necessary with ureteral catheterism to give accurate quantitative results: first, the catheter of Albarran with the terminal opening should be used, whether the catheterism is unilateral or bilateral; second, Albarran's method should be employed of methylene blue injection into the catheter and its non-appearance in the bladder excludes extra-catheter flow.

This procedure was not carried out in all of the cases, and this possible inaccuracy may modify my conclusions. I have, however, proceeded as if the results in collecting all of the urine from either kidney were accurate.

2. As to polyuria or oliguria, caused by the instrumentation, Albarran notes that with ureteral catheterism either one or the other may occur, but that when either does occur, it is *bilateral*, and believes that if the examination is prolonged for two hours that the occurrence of polyuria or oliguria is rare.

Albarran believes that the Luys' Separator causes oliguria much more frequently than does ureteral catheterism. My own work would indicate that this is to be doubted, and it is possible that it causes oliguria even less often than does ureteral catheterism.

For the accuracy of our work, however, the main point is that when either oliguria or polyuria does occur that it is *bilateral*, and therefore not influencing the comparative results of the two sides.

3. Collection of urines at inappropriate times. Albarran has proved that ureteral catheterization, following the taking of diuretics, or within two hours after meals, gives distorted results. He says " . . . . in these different circumstances" — i. e., ureteral catheterrization following diuretics or within two hours after meals — "the healthy kidney, or comparatively healthy kidney, produced temporarily much more urine, while in the same space of time the diseased kidney modified but little its habitual function."

2. THE VALUE OF THE COMPARISON OF THE TOTAL UREA EXCRETED BY EACH KIDNEY IN ESTIMATING THE FUNCTIONAL CAPACITY OF THE KIDNEYS.

Caspar (5) has made an interesting comparison of the deaths occurring after nephrectomy among the cases of five surgeons who worked

with the aid of ureteral catheterism and functional diagnosis; and those of five surgeons who did not, or could not, employ those aids, since at the time of the operation they did not exist. Of 129 nephrectomies on the side of the old school, five deaths occurred from renal insufficiency (5.22 per cent). On the side of the new school, in 129 nephrectomies one death occurred from renal insufficiency (1.7 per cent). This shows a remarkable decrease in mortality from renal insufficiency, which in part should be attributed to improved surgical technique, but chiefly, I believe, to a more intimate knowledge of the condition and functional capacity of the two kidneys.

Before discussing the pathological cases, I wish to consider briefly the total N. excreted by the two kidneys in more or less normal cases, and to compare it with the phloridzin test. I have taken from the book of Caspar and Richter, 8 cases which are called by the authors "normal cases." These are:

No.	Diagnosis	Sugar Per cent	Quant. Urine c.c.	N. Per cent	Total N. In Gms.
1.	Prolapse Urethra	R. 0.9	28	.399	.110
		L. 0.8	22	.445	.097
2.	Pyelitis Stone, Gall-stone	R. 0.4	25	.224	.056
		L. 0.4	35	.35	.122
3.	Contracted Bladder	R. 1.4	36	.213	.086
		L. 1.4	35	.206	.072
4.	Hysteria	R. 0.8	30	.357	.107
		L. 0.9	29	.413	.110
5.	Cr. Urethritis	R. 1.8	23	.616	.141
		L. 1.8	21	.616	.129
6.	Cr. Urethritis	R. 1.6	35	.219	.171
		L. 1.5	21	.448	.094
7.	Sex Neurasthenia	R. 2.	22	.525	.115
		L. 2.	20	.538	.107
8.	Neurosis Vesical	R. 0.7	35	.252	.089
		L. 0.15	42	.202	.084

In these cases the phloridzin test gave the same functional value for both kidneys in all of the cases except "Case 8," where the sugar excreted from one side is nearly five times as great as that excreted from the other, yet the diagnosis in this case is vesical neurosis. The total nitrogen of both sides in this case is equal Caspar believes that the result of the phloridzin test in this case is a mistake.

There are 2 cases in which the total N. of either side is not equal. One was a case of kidney-stone in which the diseased kidney excreted a little over twice as much total N. as its fellow; while the sugar excreted was the same on both sides. This was probably a case of pelvic-stone, and as discussed in Part III. of this article, the diseased kidney was really excreting more N. than its fellow.

The other case, which gives the total N. nearly twice as much on one side as the other, was a case of urethritis. In this case the sugar output was practically equal.

As a whole, the total N. seems to give a more accurate estimate of the kidney function than the phloridzin test. In two cases the phloridzin test was not as accurate as the total N. test. In one case it was more accurate. In one normal case the phloridzin variation was nearly five times as much on one side as the other; while the greatest variation of the total N. in a normal case was not quite twice as much on one side as the other.

The Total Urea Excretion in Pathological Cases.

The sources of the 78 cases upon which my article is based are: Albarran (6), Luys (7), Lichtenstern (8), Young (9), Vale (10), Caspar and Richter (11), Cathelin (12), Beer (13), and cases of my own observation. In all of these cases the result of the operation has been determined.[1]

In these 78 cases I have tabulated the following:

1. The instrumentation: Ureteral catheterism or urinary separation.
2. The quantity of urine excreted by the two kidneys during the same period of time.
3. The urea in grammes per litre excreted by each kidney.
4. The total urea of each kidney in grammes. (By this is meant the *total urea* excreted during the time the instrumentation was taking place.)
5. The diagnosis.
6. The operation which with one exception has been nephrectomy.
7. The result of the operation. But 2 of the 78 cases died from renal insufficiency.
8. The condition of the nephrectomized kidney as far as mentioned.

[1] In all of those cases with the exception of those taken from Albarran, the result of the operation is mentioned. The cases of Albarran are only those where nephrectomy for tuberculous kidney has been performed, and while the result is not mentioned, Caspar quotes Albarran as never having had a death from renal insufficiency following nephrectomy for tuberculous kidney. (CASPAR, loc. cit.)

I have divided the series of 78 cases into two tables, as follows:

Table 1. Those cases where the diseased kidney excreted *no* urea. Table 2. Where the diseased kidney excreted quantities of urea from a barely appreciable amount up to four or five times as much as that excreted by the normal kidney.

Table 1. This consists of 16 cases. [Cases 1 to 16 in table.] In all of the cases there was no urea excreted from the diseased kidney. In 3 of the cases there was a small quantity of fluid excreted from the diseased side but this contained no urea.

In 9 of the cases the quantity of urine or the percentage urea excreted by the normal side is not mentioned; instead is mentioned "normal urine from the healthy side," etc.

Nephrectomy and recovery is tabulated in all of the cases.

In 2 of the cases the condition of the nephrectomized kidney is not mentioned. In 9 cases the nephrectomized kidney was entirely destroyed; in 5 cases "almost destroyed."

In considering this class of cases we must be careful to exclude those cases where the ureter is temporarily blocked by pus or a stone; or where the examination is made during the crisis occurring with intermittent hydronephrosis. These conditions can almost always be diagnosed by a careful consideration of the symptoms. In these cases there may be nothing excreted from the diseased kidney, while this kidney may be practically normal.

It is self-evident that if the diseased side excretes nothing that this kidney has *no* functional value; and to say that such a kidney is "almost" i. e., partially, destroyed, as is tabulated in four of the cases, is a misnomer.

We may conclude, therefore, that in those cases where no urea is excreted from one side, and where normal, or practically normal, urine flows from the other, the kidney which excretes urine is functionating normally and is already doing the total body work, and according to the duration of the process has undergone more or less compensatory hypertrophy, and is entirely capable of taking up the total body work after the removal of the diseased kidney. [An exception to this is mentioned later.] On the side which excretes nothing the functionating power of that kidney is entirely lost. In cases in which the disease in the kidney is a pyogenic process, the normal kidney, in addition to the normal body work, may be excreting toxins occasioned by the presence of the diseased kidney.

This is frequently seen when a toxic nephritis of the more normal side disappears after nephrectomy.

Table 2. [Cases 17 to 78 in table.] These cases I originally sought to divide into two classes:

1. Where the diseased kidney excreted an appreciable amount of urea, but had not a normal functional capacity.

2. Where the diseased kidney had a normal functional capacity.

It seems impossible to indicate the division line between the above two classes without considering a larger number of cases in which death from renal insufficiency has followed nephrectomy. My series includes but two such cases. I believe, however, that a kidney that excretes one-fourth, or less, urea than its fellow has not a normal functional capacity. Therefore, *if we remove a kidney that excretes four times as much urea as its fellow, we may fear death from renal insufficiency.*

My reason for fixing this approximate limit will be discussed under the "borderland class."

I will therefore discuss these 62 cases as a whole. In these 62 cases nephrectomy was performed 61 times, and nephrotomy once. The case (Case 78) in which nephrotomy was performed will not be included in the series, but will be reserved for special discussion later.

In these 61 cases the *quantity of urine*, *per cent urea*, and *total urea* are as follows:

Oliguria of diseased kidney, 36.

Polyuria of diseased kidney, 23 (1 death from renal insufficiency).

Equal secretion, 3.

Decreased per cent urea diseased kidney, 59.

Increased, 1. (Death in this patient from renal insufficiency. Same patient as above.)

Equal urea, 1.

Decreased total urea, diseased kidney, 56.

Increased total urea, diseased kidney, 5. (One death from renal insufficiency. Same patient as above.)

The one death from renal insufficiency occurred in the series that shows polyuria, increased per cent urea, and increased total urea of the diseased kidney.

Albarran has said, "When the kidney which excretes the more urine presents at the same time higher (Δ) and more urea and phosphates by litre, that kidney ought to be considered the better of the two, not only from a functional but from an anatomical standpoint." Yet in 18 of my 23 cases of polyuria of the nephrectomized kidney this kidney excreted less total urea than its fellow. In 3 of these 18 cases the diseased side excreted three times as much urine as the normal, but less total urea. In 2 of the cases (cases 27 and 30) there was a slight polyuria of the diseased side, and these kidneys were practically non-functionating.

We may therefore add to the statement of Albarran and say that the kidney which excretes the more urine may be functionally and anatomically the poorer of the two, if the per

No	Instrumentation	Quant. Urine		Urea in Grams Per Litre		Total Urea in Grams		Diagnosis	Operation	Result	Condition of Nephrectomized Kidney
		K. D.	K. H.	K. D.	K. H.	K. D.	K. H.				
1	Luys' Sep.	0 c.c.	14.5cc	0	23.05		.333	Tbc. Kidney	Nephrec.	Recovery.	Kid. almost destroyed.
2	Luys' Sep.	0 c.c.	6	0		not	ment.	Pyonephrosis	Nephrec.	Recovery.	Kid. destroyed.
3	Luys' Sep.	0 c.c.	not ment.	0		not	ment.	Tbc. Kidney	Nephrec.	Recovery.	Kid. destroyed.
4	Luys' Sep.	0 c.c.	"	0		not	ment.	Tbc. Kidney	Nephrec.	Recovery.	Kid. destroyed.
5	Luys' Sep.	0 c.c.	"	0		not	ment.	Neoplasm	Nephrec.	Recovery.	Kid. destroyed.
6	Catheterism	0 c.c.	740	0	23.7		17.53	Tbc. Kidney	Nephrec.	Recovery.	Kid. destroyed.
7	Catheterism	0 c.c.	275	0	5.76		1.56	Tbc. Kidney	Nephrec.	Recovery.	Kid. destroyed.
8	Luys' Sep.	2 drps.	8 c.c.	0	15.93		.127	Pyonephrosis	Nephrec.	Recovery.	Kid. destroyed.
9	Luys' Sep.	1 drp.	6 c.c.	0	13.50		.081	Tbc. Pyonephrosis	Nephrec.	Recovery.	Kid. destroyed.
10	Luys' Sep. and Catheterism. Results same	0 0	not ment.	0				Pyonephrosis	Nephrec.	Recovery.	Kid. destroyed.
11	Luys' Sep.	2.3	17	0	21	0	.357	Hydronephrosis	Nephrec.	Recovery.	Kid. almost destroyed.
12	Catheterism	0	11	0	28	0	.308	Sclerosed Kid.	Nephrec.	Recovery.	Kid. almost destroyed, small amt. urine through sinus in side.
13	Luys' Sep.	0	not ment.	0				Tbc. Kid.	Nephrec.	Recovery.	Kid. almost destroyed.
14	Luys' Sep.	0	"	0				Pyonephrosis	Nephrec.	Recovery.	Kid. partially destroyed.
15	Luys' Sep.	0	"	0				Hydronephrosis	Nephrec.	Recovery.	
16	Luys' Sep.	0	"	0				Hydronephrosis	Nephrec.	Recovery.	
17	Luys' Sep.	1 c.c.	10.05	1.26	16.39	.0012	.1637	Hydronephrosis (Calculus)	Nephrec.	Recovery.	Kid almost destroyed.
18	Luys' Sep. 1st Sep. 2nd Sep. 3rd Sep.	 5 6.75 6	 3 7.5 46	 2.89 1.31 1.28	 3.75 13.50 18.91	 .014 .008 .007	 .012 .101 .869	Pyonephrosis Tbc.	Nephrec.	Recovery.	Kid almost destroyed.
19	Luys' Sep. 1st Sep. 2nd Sep.	 3 5.2	 65 106	 .03 1.28	 5.38 6.40	 .000 .006	 .349 .678	Cr. Diffuse Nephritis	Nephrec.	Recovery.	Complete Atrophy Kid.
20	Luys' Sep.	3	8	1	25	.003	.2	Pyonephrosis Tbc.	Nephrec.	Recovery.	More than ½ destroyed.
21	Luys' Sep.	¾	12	3	25	.002	.3	Pyonephrosis Tbc.	Nephrec.	Recovery.	Kid. prac. destroyed.
22	Catheterism	10	150	1.24	13.03	.012	1.95	Pyonephrosis Tbc.	Nephrec.	Recovery.	
23	Luys' Sep.	1.5	8	.82	13.15	.001	.105	Tbc. Kidney	Nephrec.	Recovery.	Kid. destroyed.
24	Catheterism	25	130	3.80	18.80	.095	2.44	Tbc. Kidney	Nephrec.	Recovery.	
25	Catheterism	315	1080	2.40	11.10	.756	11.98	Tbc. Kid., Cavernous	Nephrec.	Recovery.	
26	Catheterism	7	50	14.28	17.93	.099	.895	Tbc. Pyonephrosis	Nephrec.	Recovery.	
27	Luys' Sep.	4.5	2.5	0.75	12.10	.003	.03	Tbc. Pyonephrosis	Nephrec.	Recovery.	Kid. one half destroyed.
28	Luys' Sep.	2.5	7	5.12	33.3	.012	.233	Tbc. Kidney	Nephrec.	Recovery.	Kid. one half destroyed.
29	Luys' Sep.	3	11	5.12	14.09	.015	.154	Pyonephrosis (Calculus)	Nephrec.	Death peritonitis	Kid. almost destroyed.
30	Catheterism	58	54	1.92	17.93	.111	.968	Tbc. Kid. with corsious foci	Nephrec.	Recovery.	
31	Luys' Sep.	1.5	2.5	3	35	.004	.087	Pyonephrosis, Calculus	Nephrec.	Recovery.	Kid. prac. destroyed.
32	Catheterism	4	18	7.56	18.91	.030	.340	Tbc. Kid., Cavernous.	Nephrec.	Recovery.	
33	Luys' Sep.	3.5	7	5.12	28.62	.017	.20	Pyonephrosis, Tbc.	Nephrec.	Recovery.	
34	Catheterism	2	8	5	12.60	.01	.10	Tbc. Kid.	Nephrec.	Recovery.	
35	Catheterism	80	700	5.20	11.60	.416	8.12	Tbc. Kid., Cavernous.	Nephrec.	Recovery.	
36	Catheterism	23	123	5.60	8.10	.128	.996	Tbc. Kid., Cavernous.	Nephrec.	Recovery.	
37	Luys' Sep.	3	11	7.68	16.65	.023	.183	Tbc. Kid.	Nephrec.	Death, tbc. meningitis	Kid. partly destroyed.

38	Luys' Sep.	not ment.	3xKD	4.55	12.88	.004	.038	Tbc. Pyelonephritis.	Nephrec.	Recovery.	Kid. one half destroyed.
39	Catheterism 1st 2nd	 150 150	 300 290	 0.80 5.	 30.30 24.60	 .12 .75	 9.09 7.134	Tbc. Kid. with cavities.	Nephrec.	Recovery.	
40	Catheterism	7	24	4.48	5.12	.031	.122	Tbc. Kid. with cavities.	Nephrec.	Recovery.	
41	Catheterism	1.5	6	2.56	16.65	.003	.099	Pyonephrosis.	Nephrec.	Recovery.	Kid. destroyed.
42	Catheterism	2.5	2.5	3.72	21.09	.009	.052	Tbc. Kid. with cavities.	Nephrec.	Recovery.	
43	Catheterism	3	8	0.65	12.	.001	.096	Tbc. Kid. with cavities.	Nephrec.	Recovery.	
44	Catheterism	4	2.5	0.70	12.10	.002	.030	Tbc. Kid. with large cavity.	Nephrec.	Recovery.	One half Kid. destroyed.
45	Luys' Sep.	2½	3	6	17.2	.015	.056	Tbc. Pyclonephritis.	Nephrec.	Recovery.	Tbc. not far advanced.
46	Catheterism	750	850	4.34	11.77	3.25	10.	Tbc. Kid.	Nephrec.	Recovery.	
47	Catheterism	440	360	5.20	19.50	2.30	7.02	Rupture of Kid.	Nephrec.	Recovery.	Kid. much sclerosed.
48	Catheterism	39	10	2.50	27.10	.097	.271	Hydronephrosis.	Nephrec.	Recovery.	Kid. nearly atrophied.
49	Luys' Sep.	19.5	29.5	8.32	15.37	.162	.453	Tbc. Kid. (miliary).	Nephrec.	Recovery.	Kid. partly destroyed.
50	Luys' Sep.	5.80	12.30	11.52	15.65	.066	.192	Hydronephrosis.	Nephrec.	Recovery.	Kid. slightly affected.
51	Catheterism	32	55	11.53	16.65	.368	.915	Tbc. Kid. with cavities	Nephrec.	Recovery.	
52	Catheterism	25	25	9.92	27.30	.247	.682	Tbc. Kid. with cavity.	Nephrec.	Recovery.	
53	Catheterism	8	10	16.62	35	.132	.35	Tbc. Kid. with cavities.	Nephrec.	Recovery.	
54	Catheterism	180	67	2.40	9.40	.432	.629	Tbc. Kid. (miliary.)	Nephrec.	Recovery.	
55	Luys' Sep. 1st 2nd	 18 7.5	 10 6	 5.12 14.09	 17.93 28.18	 .092 .105	 .179 .169	Pyonephrosis Calculus.	Nephrec.	Recovery.	Kid. one half destroyed.
56	Luys' Sep. 1st. 2nd.	 5.5 7	 9 11	 5.12 6.40	 7.68 7.68	 .028 .044	 .069 .084	Tbc. Kid.	Nephrec.	Recovery.	Kid. slightly affected.
57	Luys' Sep.	5	4	7.5	16.39	.037	.065	Tbc. Kid.	Nephrec.	Recovery.	
58	Luys' Sep.	1.5	2.1	8.25	14.55	.012	.030	Cancer Kid.	Nephrec.	Recovery.	One-half Kid. destroyed.
59	Luys' Sep.	4	5	12.23	15.26	.048	.076	Tbc. Kid.	Nephrec.	Recovery.	
60	Luys' Sep.	27	17	7.30	21.16	.197	.359	Hydronephrosis.	Nephrec.	Recovery.	Enormous Hydronephrosis
61	Catheterism	600	900	11.4	11.67	6.84	10.50	Hydronephrosis.	Nephrec.	Recovery.	
62	Catheterism	800	430	6.30	20	5.04	8.60	Tbc. Kid. with large cavity.	Nephrec.	Recovery.	
63	Catheterism 1st 2nd	 100 9	 48 5½	 7.05 11.52	 14.07 28.02	 .705 .103	 .675 .154	Tbc. Kid. with cavities.	Nephrec.	Recovery.	
64	Catheterism	650	220	5.10	21.80	3.31	4.79	Tbc. Kid.	Nephrec.	Recovery.	
65	Luys' Sep.	14	13	17.29	27.54	.242	.358	Uro-pyo-nephrosis.	Nephrec.	Recovery.	Kid. partly destroyed.
66	Luys' Sep.	5	4	8.96	12.81	.044	.051	Fibromas (small) of Kid.	Nephrec.	Recovery.	
67	Luys' Sep. 1st 2nd	 5 5.5	 8 9	 2.52 3.20	 32.78 20.46	 .126 .176	 .262 .184	Pyonephrosis Tbc.	Nephrec.	Recovery.	Much of Kid. destroyed.
68	Luys' Sep.	5.2	1.5	2	8	.010	.012	Pyonephrosis (calculus).	Nephrot. Later Nephrec.	Recovery. Recovery.	
69	Catheterism	1650	1100	8.35	13.50	13.77	14.85	Tbc. Kid.	Nephrec.	Recovery.	Beginning Tbc.
70	Catheterism	18	20	3.78	3.78	.068	.075	Pyonephrosis, Tbc.	Nephrec.	Recovery.	
71	Catheterism	8.1	3.7	5.3	13	.042	.048	Pyelonephritis.	Nephrec.	Recovery.	
72	Catheterism 1st 2nd 3rd	 12 26 28	 6 12 17.5	 14.10 15.35 7.70	 25.10 18.60 12.70	 .169 .399 .215	 .150 .223 .222	Tbc. Kid. with small cavities.	Nephrec.	Recovery.	
73	Catheterism	144	60	7.05	16.60	1.015	.996	Tbc. Kid. (beginning).	Nephrec.	Recovery.	Beginning Tbc.
74	Catheterism	15	5	5.04	10.08	.075	.050	Tbc. Kid. with cavities.	Nephrec.	Recovery.	Kid. not markedly affected.
75	Catheterism	75	42	12.81	17.29	.960	.726	Tbc. Kid. with small cavity.	Nephrec.	Recovery.	
76	Catheterism	90	30	7.56	12.61	.680	.378	Tbc. Kid. with cavities.	Nephrec.	Recovery.	
77	Luys' Sep.	7	4	25	10	.175	.040	Tbc. Kid.	Nephrec.	Death	Patient died on third day after operation from renal insufficiency.
78	Catheterism	22	6.6	5	22	.11	.145	Pyonephrosis, calculus.	Nephrot.	Death from renal insufficiency	Both Kid. diseased. K. D. probably the better Kid.

Cases 1 to 16 are those where one kidney excreted no urea and the other kidney was doing the total body work. All recovered from nephrectomy.

Cases 17 to 44 are those where the diseased kidney excreted one fourth or less urea than its fellow, and probably in all cases did not have a normal functional capacity. Nephrectomy in all cases. No deaths from renal insufficiency.

Cases 45 to 72, where the diseased kidney excreted from an equal amount to one fourth less urea than its fellow. Nephrectomy in all cases. No deaths from renal insufficiency. The removed kidney had in many cases a normal functional capacity.

Cases 73 to 77, where the diseased kidney excreted more urea than the presumably normal kidney. Nephrectomy in all cases; death from renal insufficiency in but one, in Case 77, where the diseased kidney excreted four and a half times the urea of its fellow.

Case 78, both kidneys diseased. Death following nephrotomy of kidney excreting less urea.

K. D = Diseased kidney.
K. H. = Healthy kidney.
Luys' Sep. = Luys' Separator used.
Catheterism = Ureteral catheterism.

cent urea varies so as to make the total urea less on that side.

The Per Cent Urea versus the Total Urea in Estimating the Kidney Function

It might be here interjected that the per cent urea would give us a more adequate idea of the kidney function than the total urea, as in only one case was the percentage urea of the diseased side greater than that of the normal, and this case died of kidney insufficiency. While in 5 cases of the 61 the total urea was greater on the diseased side and only one of these died of kidney insufficiency.

A closer view of the two cases that died of insufficiency, one after nephrectomy and one after nephrotomy, will throw some light on this:

CASE 77. Death from uræmia on third day after nephrectomy.

In this case the per cent urea and the total urea of the two kidneys was as follows:

Per cent urea diseased kidney, 2.5 per cent.
Per cent urea normal kidney, 1. per cent.
Total urea diseased kidney, 17.5 centigram.
Total urea normal kidney, 4. centigram.

The per cent urea was two and a half times more on the diseased side than on the normal, which would not of itself contraindicate nephrectomy. But the total urea was nearly four and a half times greater on the diseased side. With one kidney doing four and a half times as much work as its fellow, the question would immediately arise as to the advisability of removing such a kidney.

CASE 78 is of much interest. The case was one of double renal pelvis and bifid ureter (14): Ureteral catheterism gave in 1 hour and 10 minutes.

R. Urine	*L. Urine*
22 c. c.	6 c. c.
Cloudy	Clear
Sp. gr., 1003	Sp. gr., 1015
Neutral	Acid
Albumen, trace	Albumen, none
Urea, 5 gr. to litre	Urea, 22 gr. to litre
Pus (microscopical)	Rare red blood cell.

The urine from the left side was normal.

"Radiograph" shows a large calculus on right side.

Diagnosis before operation: Left kidney healthy, operation advised for calculus of right kidney.

OPERATION

Nephrotomy, extraction of large stone from R. renal pelvis. Death two days later from anuria. Autopsy gave L. kidney at a point about 2 c.m. above iliac vessels, the L. ureter divided into two, which two ureters continued separate up to two separate pelves in the kidney.

The lower pelvis, which was the larger of the two, was supplied by kidney tissue, which was apparently normal in every way.

The upper portion of the kidney and its pelvis contained a large calculus, which completely filled the pelvis. The renal cortex was very thin.

In this case, while only nephrotomy was done, it was done under the supposition that the left kidney was normal. The percentage urea of the kidney upon which the nephrotomy was performed was 5 gr. to litre, while that of the kidney with the bifid ureter was 22 gr. to litre, or nearly five and a half times as much.

Yet the ratio is different when the total urea is considered, 11 gr. from the nephrotomized side, and 14.5 gr. from the bifid ureter kidney, *or a nearly equal excretion of urea.*

If our contention is right, that the total urea gives a more adequate idea of the functional capacity than the per cent urea, then the two kidneys should have been practically equal in function. And this was probably the case as the pathological findings at autopsy showed.

The Borderland Class

What cases shall we include in the borderland class is perhaps the most important question of all and the most difficult to answer.

I have said before that it is probable that a kidney that excretes one-fourth or less urea than its fellow has not a normal functional capacity. And that if we remove a kidney that excretes four times as much urea as its fellow, we may fear death from renal insufficiency.

One of the reasons for fixing this limit is that in all cases where the diseased kidney excretes one-fourth as much urea as its fellow, or less — where the condition of the diseased or extirpated kidney is mentioned — the pathological reports indicate that the kidney was nearly or wholly destroyed.

Of course, even a rough estimation of the functional kidney capacity by a macroscopical or even microscopical examination of such a kidney is a questionable procedure.

Also there are in the series 5 cases in which nephrectomy was performed in which the total urea of the nephrectomized kidney was more than that of the remaining kidney. One of these cases where the ratio of the urea of the diseased kidney to the urea of the remaining kidney was $4\frac{1}{2}$ to 1 died from uræmia following

nephrectomy. The four other cases recovered after the nephrectomy. In these 4 cases the highest ratio of the total urea of the diseased side to that of the normal was practically 2 to 1. These 5 cases would seem to establish the fact that if the ratio of the urea of the diseased kidney to the urea of the normal one was greater than 2 to 1 a borderland case was being dealt with.

On the other hand, in Case 78 the total urea of the diseased kidney was less than that of the normal, and death from kidney insufficiency followed a simple nephrotomy.

This would indicate that there must exist other indications of an impending insufficiency than the simple comparison of the work of the two kidneys. In this last case the indication was probably the low 24-hour urea — 4.95 grams.

In every case there are three things that should be considered in addition to the comparison of the work of the two kidneys. These are: the 24-hour excretion of urea, or N.; any symptoms of uræmia; and the general surgical condition of the patient.

I believe that we can always tell the comparative value of the work done by each kidney, but there exists a small — fortunately very small — per cent of cases where if even one kidney excretes nothing at all we cannot on that basis alone perform nephrectomy with impunity. Most of this small percentage of cases indicate that nephrectomy would be dangerous, either by presenting symptoms of uræmia, or by excreting for a period of time a low 24-hour urea, or nitrogen.

On the other hand, I believe there exist a very few cases where none of our known tests can establish the fact that kidney insufficiency is impending.

Why one kidney, diseased, and excreting an appreciable quantity of pus, should excrete more urea and have a better functional capacity than its fellow kidney, which excretes practically normal urine, is an interesting question. It is possible that the disease present in one kidney by an irritant action increases its functional capacity; while it at the same time exerts a depressant effect upon the work, or more properly the "reserve force," of its fellow kidney.

The 24-hour Excretion of Urea.

Our knowledge of the nitrogen excreted in the urine and its distribution among urea and the other nitrogenous constituents, dates from the publication of the paper by Folin, "Laws Governing the Chemical Composition of Urine," in 1905. Among other conclusions, Folin says, " . . . . it has been shown that kreatinin, ammonia, uric acid, and the non-determined nitrogenous residue all become relatively more and more important as the total nitrogen in the urine decreases." ". . . . urea (is) the only nitrogenous substance which suffers a relative as well as an absolute diminution with diminution in the total protein metabolism."

With normal persons, the total N., as well as the urea, has wide variation, depending upon a diet nitrogen-rich or nitrogen-poor.

It would seem that there ought to be a low limit to the 24-hour urea or better 24-hour N. excreted, which would be a contraindication to nephrectomy.

The total 24-hour urea in Case 78 — death following nephrotomy — as computed from the separate specimens of urine which took 1 hour and 10 minutes to collect, was 4.956 grams.

On the other hand, one of Folin's normal cases, which was living on a nitrogen-low diet, excreted in 24 hours but 3.47 gm. urea.

I have the record of one case in which nephrectomy was performed, and in which the total 24-hour urea prior to nephrectomy was approximately 5.6 gm. The kidney which was removed excreted practically one half of the total amount of urea.

From these few examples we must conclude that the 24-hour urea may fall very low and yet mean nothing. Further work in this line is necessary before any deductions may be drawn. For instance, accurate measuring of the food taken, and estimation of both the total N. and urea from the two kidneys.

The following views of Von Noorden are of interest. In discussing contracted kidney he observes, "Certain irregularities," (in the excretion of urinary products) "are observed; we may find, for instance, that the excretion of urea, etc., fluctuates considerably fairly early in the disease, so that in this respect the urinary findings differ from those in a normal subject

fed on the same diet. At the same time we never encounter a serious retention of the excrementitious substances. If a certain proportion of excretory products is retained on one day we will usually find that it is eliminated on the next day, or the second day following." " . . . . Whenever I have found that considerable quantities of urea, phosphoric acid, etc., were retained for long periods of time (not only for one, two, or three days), I have always noticed that the disease soon took an unfavorable turn."

It is probable that this principle of Von Noorden's, if applied to surgical diseases of the kidneys, will give us an important point of view as to a failing kidney function.

The less the total urea excreted by a diseased kidney as compared with that excreted by the normal kidney, the farther we diverge from the borderland class when the total urea is low.

Conclusions

1. In normal cases the total N. comparison gives a more accurate estimate of the functional kidney capacity than the phloridzin test, the greatest variations in the few normal cases examined being not quite 2 to 1.

2. In pathological cases: in those cases in which no urea is excreted from one side, and normal, or practically normal urine from the other, and in which acute or recent blocking of the ureter or intermittent hydronephrosis may be excluded, we may say that the kidney which excretes urine is functionating normally and is already doing the total body work; and, according to the duration of the process, has undergone more or less compensatory hypertrophy and is entirely capable of taking up the total body work after removal of the diseased kidney. In the kidney which excretes nothing the functional power is entirely lost.

3. With the total urea excreted from the diseased kidney less than one-fourth of that excreted by its fellow, we may diagnose grave disease of the affected side and a function of the opposite kidney sufficient to do the body work alone.

4. The total urea of the diseased side may be twice as much as that of the more normal side, and yet nephrectomy may be undertaken without fear of death from renal insufficiency.

5. If the total urea of the diseased side be four times as much as that of the more normal side, then there is grave fear of death from renal insufficiency following nephrectomy. The borderland cases are those where the total urea of the diseased side is more than twice and less than four times that of the well side.

6. The 24-hour excretion of the urea or N. should be considered in all cases. The nearer the total urea of one kidney approximates that of the other, the more importance does a low 24-hour excretion of urea or N. assume. The continued retention of urea or N. will probably indicate a danger point.

7. The general condition of a patient should be carefully considered and any uræmic symptoms sought for.

8. The comparison of the total urea to determine the functional capacity of the kidneys is better than the percentage urea.

9. Irrespective of the amount of work done by either kidney, there exists a very small class of cases where any known tests cannot establish the fact that death from kidney insufficiency is impending. These cases will be revealed, I believe, by a further study of the elimination of the excretory products of the kidneys.

3. THE VALUE OF THE COMPARISON OF THE TOTAL UREA EXCRETED BY EACH KIDNEY AS INDICATING THE PATHOLOGICAL CHANGES OF THE KIDNEYS.

I shall not go into this subject in detail, but simply mention some observations that are of clinical interest:

Unilateral nephritis. Under this class I have 6 cases, including 1 of hæmorrhagic nephritis; 4 of the cases are of my own observation and 2 from the series of Luys.

In these cases the appearance of casts, granular, epithelial, or the pathological examination of specimens excised from one kidney at operation, have caused the diagnosis of unilateral nephritis to be made.

In these cases there was a slight polyuria of the affected side in 1, and oliguria in 4, and equal excretion in 1. The percentage urea is given in but 4 of the cases, and in these 4 the total urea is less on the affected side; and the diminution seems to follow the extent of the pathological lesion.

It is interesting to note that a unilateral nephritis, even if it has existed for a number of years, does not cause a marked polyuria of the affected side. In one case of hæmorrhagic nephritis, which had existed eight to nine years, the functional capacity of the two kidneys was practically equal.

Kidney-stone. I wish to speak of but one important point; that is the polyuria often present with calculus of the renal pelvis. Albarran notes that with marked calculus pyelonephritis we may have a notable polyuria of the affected side, and quotes the following case:

"Enormous calculus of pelvis of kidney:
Healthy kidney, 37.5 cc. (Urea not given).
Diseased kidney, 68. cc. (Urea not given)."

It would seem that the calculus must be in the kidney pelvis to cause this marked polyuria, and not a simple calculus pyelonephritis. I have been unable to find a case of marked polyuria in calculus pyelonephritis if the calculus was not in the kidney pelvis.

The following example from Luys is much more remarkable than that of Albarran's:

Quantity	Urea in Grams per litre	Total Urea
First Examination.		
Healthy Kidney..... 1 c.c.	5.12	.500
Diseased Kidney.....15 c.c.	9.80	.147
Second Examination.		
Healthy Kidney ..... 3 c.c.	5.80	.017
Diseased Kidney.....34 c.c.	12.81	.435

Nephrotomy showed calculus of L. kidney pelvis. The urine from the R. side was practically normal.

One of my cases shows a moderate polyuria of the affected side.

K. H. — 9 c.c.
K. D. — 12 c.c.

Nephrotomy of K. D. showed stone of kidney pelvis. This polyuria, if it is marked, generally corresponds with a kidney of good functional capacity.

Pyonephrosis. Albarran says, "The only surgical disease of the kidneys in which we have never encountered a relative polyuria of the diseased kidney is pyonephrosis."

In our cases we have the following cases of pyonephrosis, excluding tuberculous — 4 cases in the first 16, where nothing was excreted from one side and where the kidney was practically destroyed.

In the other cases of the series there are 6, as follows:

Oliguria of diseased kidney, 3.
Polyuria of diseased kidney, 3.
Decreased total urea, diseased kidney, 6.
Increased total urea, diseased kidney, 0.

One of these three cases (Case 66) of polyuria of the diseased kidney shows 5.2 c.c. from the diseased kidney, and 1.5 c.c. from the normal kidney.

All of these cases of pyonephrosis, including those of tuberculous pyonephrosis, show the total urea of the diseased side less than that of the normal kidney.

Another case of polyuria of the diseased kidney was Case 53, where there was a large calculus of the kidney pelvis.

Conclusions. In the majority of cases there is an oliguria of the affected side. The more marked this is the more nearly is the kidney destroyed. If there is a polyuria of the diseased kidney this generally indicates that much of the kidney tissue is still present. In all of our cases the total urea of the affected side is diminished. The total urea seems to indicate accurately the amount of kidney destruction.

Tuberculous kidney. In the series there are 52 cases of tuberculous kidney as follows:

	Cases
Oliguria of diseased kidney.....	35
Polyuria of diseased kidney	15
Equal secretion	2
Decreased urea of diseased kidney	47
Increased urea of diseased kidney	5

I can add nothing to what Albarran has already mentioned: "Polyuria of the diseased kidney is especially frequent where the lesions are not yet very advanced, but it may be observed when the kidney already presents multiple caverns; in this case, although very altered, the diseased kidney yet has a notable portion of parenchyma left. Where the lesions are much more advanced, we see the healthy kidney secreting much more than the diseased."

Hydronephrosis. There are in the series 6 cases of hydronephrosis. These show:

Oliguria of diseased kidney, 4 cases.

Polyuria of diseased kidney, 2 cases.

Decreased total urea of diseased kidney, 6.

Increased total urea of diseased kidney, 0.

Albarran says, "Polyuria of the diseased side always coincides with slight lesion of that side."

This is not always true. See Case 45 — with a marked polyuria of diseased side and the kidney nearly atrophied.

There are too few cases of kidney neoplasm to make any conclusions.

Conclusions

With all kidney conditions a relative polyuria or oliguria of the diseased kidney may be present, the latter predominating. When polyuria of the diseased kidney exists, generally speaking, a less grave lesion is indicated than when oliguria is present. Polyuria, with increased total urea, seems to be most frequently seen with pelvic stone, and next in frequency in tuberculous kidney. It is possible that a pelvic stone by its perhaps irritant effect may cause both a polyuria and increased urea out of proportion to the real functional capacity of the kidney.

In all kidney conditions the comparison of the total urea excreted by either kidney seems to indicate very accurately the extent of the pathological changes in the kidneys.

I wish to express my indebtedness to Dr. E. L. Keyes, Jr., for much help and many suggestions in the preparation of this article.

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DEPARTMENT OF TECHNIQUE

TOTAL AVULSION OF THE SCALP

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Mrs. A. McR., 32 years of age, while attempting to adjust a small belt of a sewing machine to the revolving shaft in a factory, leaned too close to the machinery, and her hair, which was very heavy, became caught in the shaft. Instantly her head was brought into violent contact with the shaft, and the entire scalp, including the forehead, the right eyebrow, the occipito-frontalis and left temporal muscle, together with the right ear (except the tragus and lobule), was torn completely from the head and left dangling from the revolving shaft, whence it was beaten by a few thousand revolutions against the floor. The cranium was denuded of periosteum for a large area over the right parietal eminence and an irregular patch about the size of a dollar in the medial line at the crest of the forehead. More than half the right eyelid was pulled off, and the left was also so loosened and torn into strips that it took more than a dozen stitches to hold it in any measure together. The tear of the scalp extended well down between the eyes in front to the bridge of the nose, and down the back of the neck to about the fourth cervical vertebra, but the skin edges were undermined and loosened for a distance of some three to five inches down the neck and sides of the face. Just along the border of the flaps on the left side and for a short distance along the ragged tissue at the back of the neck the tear included the hair-line for possibly one-half inch.

Immediately upon the receipt of the injury, her head was wrapped in sterile gauze by Dr. A. C. Boswell, and she was taken to the hospital, where I saw her about an hour later. She had not lost consciousness, although the shock was severe and the loss of blood had been very great. The temporal and occipital arteries were at once secured by hæmostatic forceps and the other bleeding points controlled by pressure while the patient was placed under the influence of ether. Her pulse improved somewhat under the anæsthetic. The immense wound and the skin flaps were cleansed thoroughly and the remainder of the scalp was stitched up to the periosteum and thus held against the skull. This reduced the denuded area somewhat, but the great dome of the skull, the forehead, and the right side of the head, remained unprotected — a terrifying, and apparently, almost hopeless problem. As soon as the head was thoroughly cleansed surgically, I at once cut several large (Tiersch) grafts from the patient's thigh, and covered the forehead and right side of the head with them. By this time the condition of the patient was so critical that further attempts at grafting had to be discontinued. She, however, reacted fairly well from the anæsthetic, but as the loss of blood had been great she was given a pint or more of normal salt solution by hypodermoclysis. The wound remained absolutely aseptic. The grafts grew nicely. The remainder of the wound was dressed with a sterile ointment of balsam of Peru and vaseline. All the grafts thus applied "took," although they were placed directly upon the periosteum.

November 2, 1907, the patient was given an anæsthetic and a large amount of skin was transplanted from the inner surface of her thighs and upper arm. These grafts were applied to the upper portion of the forehead and top of the head. An effort was made only to cover one portion of the head at a time; it was found that to do more than this was useless, as the newly applied grafts were so easily displaced either in handling the head in applying the dressings, or by the patient herself rubbing them loose during sleep. These grafts also grew.

November 11, 1907, skin grafts (Tiersch), were taken from the patient's sister. As large an amount of skin was transplanted at this time as possible. These grafts were cut under the influence of a general anæsthetic. The fresh granulations were entirely removed by a curette, and the transplanted skin was applied directly to the periosteum.

November 16, 1907, grafts were taken again from the patient's sister and applied to the opposite side of the head.

November 24, 1907, skin grafts (Tiersch) were taken from the patient's brother, cutting them from the inner surface of the thigh, and applying

Showing photograph of the regenerated tissue.

them to the remaining denuded area. These last grafts covered the head completely, and all of them apparently were growing satisfactorily. Ten days after these last grafts were applied (the fortieth day), the case was presented to the Jackson County Medical Society with the whole area completely covered.

In a week or ten days after this time I noticed, however, that the grafts which were taken from the sister and brother were becoming very thin. They were apparently being dissolved, or digested by cellular activity. Although they grew nicely for from two to three weeks, about the end of this period of time they began to disappear.

On December 18, 1907, only a few of these heterogeneous grafts remained. Without an anæsthetic this time, I transplanted quite a number of Reverdin and small Tiersch grafts from the patient's arms. The skin which was transplanted from the patient's own person had, in the meantime, grown very rapidly, and had spread an inch or more beyond the places upon which it had primarily been placed.

January 1, 1908, ether was given, and enough thick Wolfe-Krause grafts were cut from the patient's thigh to cover all the remaining denuded area from which the heterogeneous grafts had been dissolved. These grafts were cut as thick as possible, the effort being made to exclude only the underlying fatty tissue. Within from two to three weeks the small patches of granulation tissue between the grafts had become completely covered with epithelium, and the case was discharged. That this was a mistake, however, very soon became apparent. In about a week the patient returned with a small area eroded on the right side of the head, and also one or two small ulcerations just above the forehead where the dressings caused too great pressure. These ulcerations healed rapidly, leaving a moveable scar; but from this time on the greatest care and watchfulness had to be exercised to prevent their recurrence. The slightest pressure would cause an exfoliation of the skin, and unless these crusts were at once cleansed or removed microbic infection took place beneath them, and soon a rather extensive superficial suppuration had destroyed even the deeper layers of the skin. This occurred less frequently in the Wolfe-Krause grafts than in the transplantation after the true Tiersch method. Realizing this fact, my later grafts were all cut as thick as possible, and in many instances there are now quite extensive patches of fine hair growing from them. These thicker grafts seemed to grow as readily as those in which only the superficial layers were transplanted. My success in this particular will cause me to attempt, at a later period, the grafting of an eyebrow.

My rather extensive experience with this case has taught me many things about skin grafting, particularly grafting on bone, and had been of no little assistance in correcting operative defects in three other cases since that time. The older teaching that grafts can be made to grow only on an area from which luxuriant granulations have been removed, has been demonstrated as erroneous. Skin grafts will grow most readily on a primary wound surface — in fact, to my mind, they will more certainly do so. Almost immediately upon placing such grafts, serum is thrown out and through the action of the fibrin or fibrinogen the transplanted tissue is made to adhere to the wound surface. Its nourishment is carried on, according to Golman ("Offene Wundbehandlung bei Hauttransplantation." Zeutralbl. f. Chir., 1906, p. 713), at first by the extension of the circulation through this plastic lymph. The organization of this material is identical with the pathology of primary union in wound repairs.

In this connection it is interesting to note that the filaments of the nerve terminals penetrate the grafts, or are in some manner reunited with those already existing in the transplanted tissue, and that sensation returns at a surprisingly early period. From the experimental researches of Burkhardt (Deutsche Zeitschrift für Chirurgie, bd. 79), it has been shown that in skin transplantation in dogs, the sensation returned in some instances in twenty-four hours. Generally,

however, not until from five to ten days, sometimes even at a later period, and that the return of sensation extends inward from the periphery. In my own case, herein reported, sensation returned in the transplanted tissue rather gradually. It extended upward from the edge of the wound, and at the present time sensation of pain, heat, and cold has extended from three to three and one-half inches, but still the great dome of the head remains insensible to pain. This fact was the cause of a rather severe infection a few weeks ago, when the patient, in pinning on her hat on top of the wig, thrust the hat-pin through the newly transplanted skin. Prompt surgical attention prevented an extension of the infection, and the wound soon healed completely.

The technique followed in this and my other cases has been simple in the extreme. The skin to be transplanted is shaved and scrubbed thoroughly with green soap and water, then with alcohol, ether, and bichlorid solution, and afterwards thoroughly rinsed with sterile water. An anæsthetic was generally given. The grafts were cut with an ordinary hollow-ground razor after cleansing the same in alcohol. No special instruments were needed but occasionally the use of an inch-wide retractor with sharp teeth was of service in steadying the skin, the operator making firm pressure just below the point of the razor with a piece of sterile gauze. The grafts can be cut in this manner an inch and a half to two inches in width and of almost any length desired. Those, however, about one and one-half inches by two inches, it was found, were the more easily handled. The razor should be kept wet by frequently having the assistant drop normal salt solution upon it from a sponge. In grafting over bone, or other hard surfaces, it is unquestionably best to cut the grafts rather thick, including, besides the stratum-corneum and the stratum-lucidum, the stratum-Malpighii, or the rete mucosum. In many of the grafts the whole thickness of the skin was taken, including the coreum proper, but all fatty tissue was carefully removed from the transplanted tissue before it was placed in position. It is well to have the skin grafts reach fully to the border of the area covered, but it is unnecessary that they should overlap, as after it is removed the graft shrinks to its fullest capacity, and when once applied to the raw surface it so soon adheres to the surface that very little contraction takes place. After the graft is applied it should be pressed firmly in position with a sponge wet in normal salt solution, and over it is placed perforated strips of rubber-protected tissue. There should be space sufficient left between these strips to afford ample drainage. Over this a layer of gauze, which has been covered with a five per cent balsam of Peru ointment, is placed, having sufficient cotton to absorb the excess of wound secretion. This dressing is changed after 48 hours, and again in two or three days, when the rubber protective can be dispensed with and a dry dressing applied. Ointments of any sort must be used with extreme care after the newly transplanted tissue has begun to grow, as they seem to macerate the skin and it very readily sloughs. Care must also be used in removing the small crusts which form over the delicate scar tissue and between the grafts. If they are removed too early an infection may result, and on the other hand if they are left any great length of time microbic infection starts in the superficial layer of the skin beneath them, and unless promptly relieved may cause extensive destruction of epithelium. Continual watchfulness of such a case is also necessary to protect the newly transplanted skin from pressure. This is particularly essential for the first few months after transplantation, as the nerve filaments have not yet completely become reorganized. After sensation has returned, however, the danger of infection from pressure is very much lessened, but a protective cap must be worn beneath the wig for a long period of time.

From the literature at my command I have been able to find but little bearing directly upon this subject of skin grafting on bone, and comparatively few cases of total avulsion of the scalp are reported.

Enz ("Zur Ueberhäutung und wund behandlung bei totaler Skalpierung des Kopfs." Korrespondenzbl. f. Schweizer Arzte, 1905, No. 22) describes a case of complete loss of scalp, including the eyebrows. The defect was covered with Tiersch flaps and healed completely without cicatricial retraction after a period of about fifteen months. Attempts to transplant flaps from the scalp removed were in almost every instance a failure. He has collected a summary of twenty-five cases from the literature, but the reports which have come to my hand have been so meager that I have been able to gain practically nothing from them.

Probably the most complete report of a case of total avulsion of the scalp is that by Dr. E. J. Mellish (The Annals of Surgery, November, 1904). In this case the scalp was entirely healed in from 70 to 75 days, and as is very aptly stated, this time could have been reduced by half by avoiding the use of heterogeneous grafts. Practically the entire head was covered with new skin in three weeks after the injury, but, as in my own

case, the grafts taken from other individuals slowly disintegrated and had to be replaced by transplantation from the patient herself.

Dr. W. Troy Bivings, of New York, also has contributed a most interesting report of a case of scalp avulsion (*Philadelphia Medical Journal*, June 7, 1902). In Dr. Bivings' case, also, all the grafts taken from sources other than the patient herself, failed to grow more than three or four weeks, when they had to be replaced. In this case attempts were made, not only to grow skin from the father and friends of the patient, but also egg skin and frog skin, and other methods of heterogeneous grafting were tried, but all failed. This case was completely covered with permanent skin in from eighty to ninety days. My own case healed in about eighty to eighty-five days.

In contrast with these comparatively recent cases, it is interesting to note the time required in many of the cases prior to the last five years. "Gussenbauer (quoted from Dr. Mellish's report) effected healing only after twenty months, owing to the continual breaking down of the cicatrices. In this case 340 pieces of skin were grafted. Nearly one-fifth of these grafts failed to 'take.'" He reports another case in which death resulted about a year after the injury. Abbe, of New York, has reported a case in which he transplanted over 12,000 grafts. Dr. Bivings, after a careful research of the literature, was unable to find more than thirteen cases of avulsion of the scalp, and some of them not complete. "Of these thirteen cases, one was reported by Hyrtl, two by Gussenbauer, two by Robertson, one by Sutliff, one by Warren (J. C.), one by Warren (J. M.), one by Lucas, one by Gerok (M.), one by Abbe, one by Sick, one case (under treatment) in Gouverneur Hospital."

Death frequently follows complete avulsion of the scalp from hemorrhage and shock. Probably infection is the greatest source of danger, causing, as it does, death from meningitis, encephalitis, erysipelas, thrombosis of one or more of the sinuses of the brain, or exhaustion from long continued suppuration. It is interesting to note that the cases which have recovered have been those in which no attempt was made to replace the completely torn-off scalp, but the defect was covered by Tiersch or Wolfe-Krause grafts taken from the patients themselves.

Heterogeneous grafts, membrane and epithelium from various animals, while occasionally growing for a time, have in every instance which I have been able to find, failed to supply a permanentlv satisfactory integument.

A CASE OF TUBERCULOUS DISEASE OF THE CŒCUM AND APPENDIX

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Mrs. S., aged 28 years, was admitted to the Presidency General Hospital, Calcutta, on November 22, 1907. She gave the following history: About seven years ago, while sitting watching a play in a theatre, she was suddenly seized with a violent pain on the right side in the lower part of the abdomen; she had been in perfect health in every respect previous to this attack, which lasted for about twelve hours and was accompanied by vomiting.

Following on this first seizure she suffered from similar attacks of pain and vomiting lasting from twelve to twenty-four hours, and occurring at intervals of between two and three weeks, for a period of about two years. At the end of this time for the space of nine months or so, beyond the occasional occurence of slight pain, she kept in good health, but at the close of this period the attacks began again as before, and have gone on increasing in frequency and severity since. From May, 1907, there has been almost continuous pain with severer exacerbations at frequent intervals.

Practically every attack has been accompanied by vomiting, and obstinate constipation has been a marked feature throughout the whole course of the illness. There has been a steady loss of weight since May, as patient has been unable to eat and drink anything without bringing on an exacerbation of the pain, and indeed previous to this she noted that after partaking of any hearty meal she always got an attack of pain.

She first noticed the swelling on the right side of the abdomen during the first attack of pain, but in the beginning it was only present during the actual attacks, growing bigger and bigger for a space of three to five minutes, and then slowly

disappearing with a gurgling sound. When first noticed it was somewhat higher up on the right side than it is at present.

During one of her earlier attacks she was admitted to hospital, and was told that her symptoms were due to disease of her ovaries and womb, for which an operation was suggested, but which she refused to undergo. Again four years ago she was admitted to another hospital where she underwent an operation for fixation of her right kidney, as undue mobility of this organ, was considered to be the cause of her trouble.

In October, 1907, she came under the care of Dr. Hill Jamieson of Penang, who recommended her to come to this hospital, and gave her a note stating that in his opinion she was probably suffering from a stricture in the region of the cœcum.

Past History. About fourteen years ago she fell a distance of about twelve feet on to her back from a verandah, but was only compelled to lay up for a few hours after the fall and appeared to have quite recovered without any after results. With this exception she has never had any other accident or illness during her life as far as she can recollect.

There is nothing of note in her family history, and none of her relations have ever suffered from any tuberculous manifestation. The husband is alive and well, and she has one son aged eleven years, quite healthy. There have been no other children, but one miscarriage at about two months pregnancy, nine years ago.

On admission the patient was very thin, emaciated, and anæmic; the tongue was furred and dry; examination of the chest revealed no abnormalities but the heart sounds were faint and distant. Abdominal palpation revealed the edge of the liver coming about three-fourths of an inch below the costal margin in the nipple line on deep inspiration, the edge was smooth and of normal consistency, the upper limit of liver dulness being the sixth rib in the nipple line.

There was a distinct sausage-shaped tumour about two and a half by one and one-fourth inches lying in the region of cœcum. During palpation of this tumour it gradually enlarged until it reached a size of about four by two and one-half inches apparently by distension of the gut with wind, and after the lapse of a few seconds it slowly diminished again to its original size with a gurgling sound as of wind passing through the bowel.

Nothing else abnormal could be detected in the abdomen. Vaginal examination revealed nothing abnormal in the pelvic contents.

Patient was in almost continuous pain, with acute exacerbations which could only be controlled by the use of morphia.

On November 26th, under chloroform, I opened the abdomen by a three-inch incision just internal to the outer border of the right rectus, the upper end of the incision being about the level of the umbilicus.

The muscle was pushed aside and the peritoneal cavity opened. A tumour was found in the cœcum, the lower few inches of the small gut were markedly hypertrophied, the walls being nearly one-fourth of an inch thick, the cœcum itself was involved in the tumour and the ascending colon was somewhat shrunken and atrophied. The appendix was much enlarged and thickened and there were several hard calcareous glands in the mesentery. A clamp was placed on the small bowel about five inches from the ileocœcal valve, and the gut divided beyond, sterilized gauze having been carefully packed all round. The proximal end of the gut was then closed with a continuous catgut suture through all the coats, and this suture was then buried by interrupted Lembert stitches also of catgut. Some difficulty was experienced in satisfactorily burying the first row of sutures, owing to the great hypertrophy of the gut.

(A second clamp had previously been placed on the ascending colon about two inches above the tumour.)

The piece of small intestine which had been divided off from the rest was now removed, together with the cœcum and appendix, by cutting through the attached mesentery, all bleeding points being picked up as they were encountered, and afterwards ligatured with fine silk. The cut end of the ascending colon was then closed in a similar manner to the small bowel.

The cut edges of the mesentery were now stitched together with the interrupted catgut suture after they had been doubled on each other, and the raw edges carefully buried with fine catgut.

The parts having been thoroughly cleansed with sterilized gauze dipped in warm saline solution, the lower end of the small bowel was placed alongside the lower end of the ascending colon and a lateral anastomosis made between the two (with an opening rather more than two inches long) by a continuous catgut suture through all the coats, this being afterwards buried by fine Lembert sutures of catgut all round. The gut was again carefully cleansed and returned into the abdominal cavity. As the operation had already lasted a considerable time and the patient was not in very good condition, and as moreover the glands all appeared to be hard and calcified, I decided that it would be unwise to further prolong the operation by any attempt at the removal of these. The abdominal walls were therefore rapidly closed with interrupted

silk worm gut stitches passing through all the layers.

During the course of the operation, as the patient's pulse was rather weak, two pints of normal saline were infused into the right median basilic vein.

Patient stood the operation fairly well but she was very noisey and restless after coming round from the effects of the chloroform and had to be kept under the influence of morphia for the remainder of that day and the following night. After this she became quieter, and one drachm of Panopeptone was ordered four-hourly from the morning of the 27th. On the evening of the 28th she had to have a further injection of morphia grains one-sixth as she was again restless and in some pain. On the 28th she was allowed albumen water two ounces, two-hourly, and the panopeptone was given two-hourly. On the morning of the 30th one-half grain doses of calomel were ordered every two hours, and after she had taken six of these the bowels were opened satisfactorily during the night. Thereafter the diet was gradually increased and on December 4th the stitches were removed and the wound was found to have healed nicely. Convalescence was uninterrupted, and she was discharged on December 31st, forty days after admission.

I saw her again on January 16th of this year; she had kept in excellent health and had a most voracious appetite. I again saw her on the 23d of January and on having her weighed, it was found that she had gained over a stone since the operation and was in robust health.

I did not see her again until June 22nd. She stated that she had had no aches or pains of any kind. She looked the picture of health, and weighed nine stone one and one-half pounds, a gain of two stone since January 16th. Her appetite, she stated, was still most voracious.

The abdominal scar was in excellent condition and there was no trace of weakness in this part.

Parts removed. About four inches of the lower portion of the ileum, together with the whole cœcum and appendix. The small gut was markedly hypertrophied, the walls being almost one-fourth inch thick. The appendix was enlarged to the size of the index finger, was about three inches in length, and considerably thickened, and the cœcum was occupied by a growth in its walls situated chiefly in the lower part; this had produced a constriction just above the ileocœcal valve almost occluding the lumen of the gut. On cutting into the growth it had somewhat the appearance of a malignant tumour. There was no ulceration of the mucous membrane in any part of the gut removed, nor were there any tubercles on the peritoneal coat.

I am indebted to Major Leonard Rogers, I. M. S., Professor of Pathology at the Medical College Hospital, Calcutta, for the following report:

"The cœcum and vermiform appendix removed by Dr. Holdich Leicester showed the following pathological changes: The wall of the cœcum was greatly thickened and its calibre much contracted. The mucous membrane showed no ulceration. The vermiform appendix was generally thickened and adherent to the back of the cœcum.

"Microscopically the wall of the cœcum was found to be extensively infiltrated with tubercle, the sub-mucous coat being much thickened and numerous giant cells being present, while the muscular coat showed scattered tuberculous infiltration. The vermiform appendix showed similar, but less extensive, tuberculous changes."

I have thought this case worth putting on record, as tuberculous disease of the cœcum is by no means common, and as in this case, at least, the diagnosis had puzzled several medical men in its earlier stages. The peculiar increase and decrease in size of the tumour when seen by me left no doubt as to its being situated in the bowel, though its exact nature was difficult to determine until the abdomen was open, and even then, at first glance, it looked extremely like a malignant growth. I do not think there is any question as to the treatment adopted being the best, the only moot point being as to the wisdom of leaving the glands behind; but my reasons for doing so are fully given above, and when glands are calcified to such an extent as in this case they are most unlikely to set up further mischief.

MALIGNANT TUMORS OF THE OVARIES[1]

Report of Eight Cases

By WILLIAM S. GARDNER, M. D.
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Malignant tumors of the ovaries are not only common, but occur in great variety. In this report of eight cases there is one hypernephroma, one hemangioendothelioma perivasculare, two carcinomata developing in adenocystomata, one developing in a papillocystoma, one adenocarcinoma which is probably metastatic, and two varieties of sarcoma.

The frequency of malignant tumors of the ovaries is variously estimated. Norris states that it varies with the different operators and gives the following table:

Olshausen, 15 per cent of all ovarian tumors malignant.

Schroeder, 16 per cent of all ovarian tumors malignant.

Fritsch, 18 per cent of all ovarian tumors malignant.

Leopold, 23 per cent of all ovarian tumors malignant.

Kelly, 8 per cent of all ovarian tumors malignant.

Freund, 21 per cent of all ovarian tumors malignant.

Werder, 25.3 per cent of all ovarian tumors malignant.

Gynecological Department, University of Pennsylvania Hospital, 15.8 per cent.

The following is a description of the tumors we have studied:

HYPERNEPHROMA OF THE BROAD LIGAMENT, PROBABLY OVARIAN

Under a strict classification of malignant tumors of the ovaries this growth should probably be excluded; but being a broad ligament tumor which in the course of its development involved the ovary to its complete destruction, and being of such an unusual variety, it was thought at least allowable to include the report with the other malignant growths of the ovaries.

Nos. 120 and 400. Miss L. M., age 49, was admitted to the City Hospital August 10, 1905; no pregnancies; has not menstruated for seven or eight years; bowels constipated; urination normal.

Fig. 1. No. 120. Hypernephroma of the broad ligament. Gross specimen. A large mass of irregular shape with numerous nodular projections. Its largest circumference is 45 cm. and its weight is 1.5 kilos. The tube is adherent to the upper border of the tumor and the fimbria are free. Numerous tortuous and congested blood-vessels are observed on the outer surface. A number of the nodules were laid open; the larger number were solid; in others a mucoid substance was found. To the naked eye the appearance of carcinoma is strongly suggested.

Her present symptoms began two years ago, when she first noticed a tumor in her abdomen which rose above the pelvic brim to the left of the median line. At first there was no pain, but as the growth developed from time to time there were severe pains originating in the pelvis and radiating in all directions. The bowels became obstinately constipated, and urination was frequent and painful; there was backache and headache; the appetite was good.

Examination. A large hard mass occupying the greater part of the lower half of the abdominal cavity and projecting into the pelvis is easily palpated. The tumor is very irregular in contour and presents a distinct nodule about the size of a hen egg on its upper anterior aspect. The abdomen contains free fluid. Through the vagina the mass is felt filling the pelvis and displacing the uterus downward.

Operation. August 11. The abdomen was opened a little to one side of the median line. The abdomen contained a large quantity of dark

[1] From the Gynecological and Pathological Departments of the College of Physicians and Surgeons, Baltimore, Maryland.

Fig. 2. No. 120. Hypernephroma. Low power.

fluid which was mopped out and the tumor exposed. Its surface was nodular and traversed by many large veins. Owing to the numerous and strong adhesions which bound it to the pelvic and abdominal structures, much difficulty was experienced in freeing the tumor. There were several quite large cysts in the growth and the walls were very friable. There was considerable hæmorrhage from the broken adhesions and from the base of the broad ligament where the attachment was strongest. The abdomen was closed without drainage.

Nothing that had the appearance of an ovary or the remnants of an ovary was discovered and it was presumed that the growth had started in the hilum of the ovary and extended into the broad ligament.

The patient convalesced rapidly and left the hospital, Aug. 30.

Miss L. M. Readmitted to the hospital, July 30, 1906, on account of abdominal pain and recurrence of the tumor.

Examination. A mass very similar to but not so large as the one removed a year ago can be felt projecting above the brim of the pelvis to the left of the median line. Her general condition is good; urine normal; blood negative.

Operation. August 1. A rounded mass which after removal measured 10 x 13 cm. was found in the region of the broad ligament on the left side. It was densely adherent to the intestines, to the pelvic wall and to the side of the uterus. When the mass was peeled out a large ragged cavity was left which oozed quite freely from many points. The larger vessels were tied but it was necessary to pack the cavity with gauze to control the bleeding from the smaller points.

August 21. The patient was discharged from the hospital.

July 30, 1908. The patient is in good health, and there are no signs of recurrence.

Histopathology. No. 120. Sections made from this case showed the following condition: The oviduct which is attached to the gross specimen is perfectly normal, and although two dozen or more sections were taken from various parts of the growth no evidence of ovarian tissue could be discovered. Whether the tumor began in the broad ligament and subsequently involved the ovary or whether the ovary was primarily the seat of origin it is impossible to say.

Sections of the capsule present nothing of interest other than a well-marked tendency to myxomatous degeneration. The peripheral portions of the growth are much denser than the central, which has the consistency of normal testicle.

Sections from the denser areas show either columns or circular islands of epithelial cells suggesting the structure of the glomerular and fascicular zones of the adrenal. The cells contain a relatively large, deeply staining nucleus and a narrow rim of oxyphilic cytoplasm. The stroma is well supplied with both blood and lymph vessels.

The sections from the more succulent portions

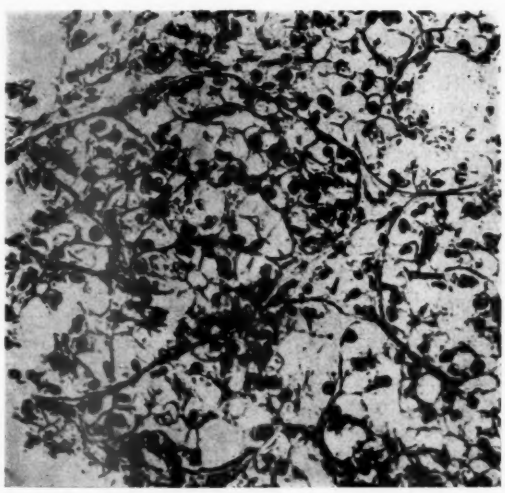

Fig. 3. No. 120. Hypernephroma. Same section as shown in Fig. 2 more highly magnified.

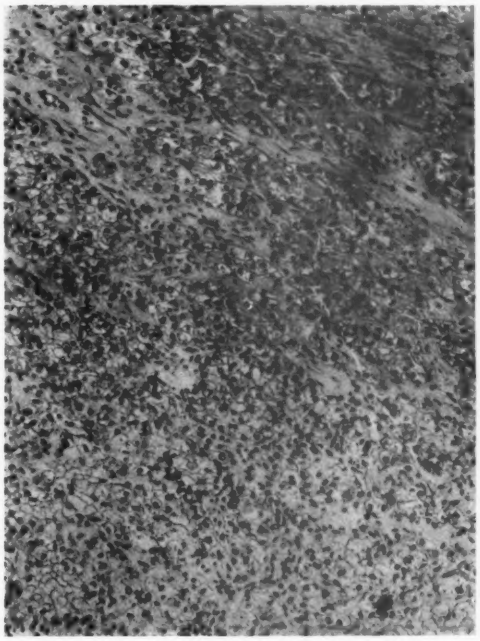

Fig. 4. No. 120. Hypernephroma. Low power.

Fig. 5. No. 120. Hypernephroma. Same section as shown in Fig. No. 4 more highly magnified.

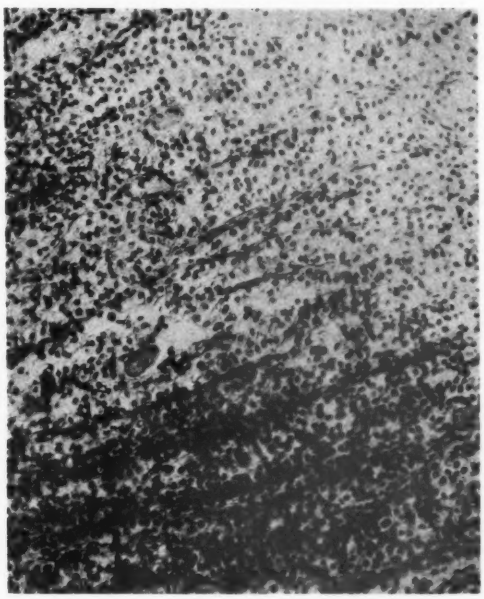

Fig. 6. No. 400. This section is very similar to the one shown in figure 2. The section was made from the tumor removed at the second operation. Low power.

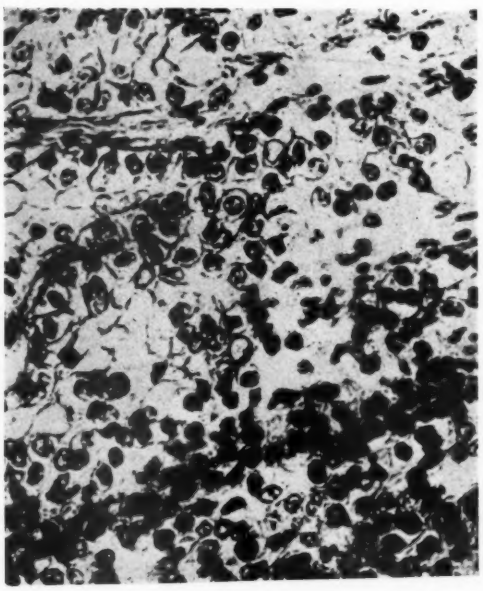

Fig. 7. No. 400. Same as figure 6 more highly magnified.

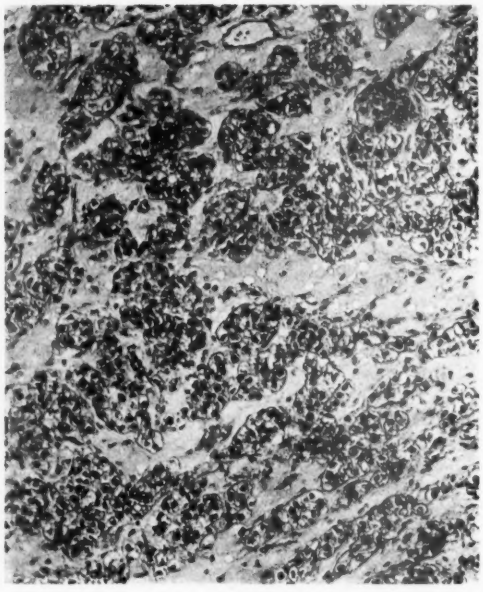

Fig. 8. No. 400. Section from tumor removed at the second operation. Low power.

of the growth and from which the illustrations are taken, present an entirely different picture. The tumor cells are deposited in well-marked alveoli which vary in size from 100 microns, to ones occupying an entire low-power field. The tissue forming the alveolar wall is fibrous, and of medium density, and from it a delicate reticulum can at times be traced into the cell groups. Both the blood and lymph vessels appear to be confined to the stroma as none could be definitely made out among the tumor cells. The tumor cells in this section are strikingly characteristic. They are large, 20 to 30 microns, polygonal, with a well-staining, centrally placed nucleus and a well marked nucleolus. The cytoplasm is very scant and shows feebly if at all, giving to the cell an empty look, except for the presence of the nucleus. Numerous cells have ruptured and the nucleus and protoplasm have escaped, leaving remains which are suggestive of soap bubbles. This same appearance is frequently observed when there is no visible sign of rupture. This picture obtains in no other neoplasm with which we are familiar and is identical with another hypernephroma of the kidney of undoubted adrenal origin in our collection. Some sections show complete necrosis of both cells and stroma — only a structureless mass remaining.

The similarity in structure of this tumor and one described by Eastwood is most striking. As far as we are able to find out there are but two hypernephromata reported, in this location, but in both cases the ovaries were intact.

Diagnosis. Hypernephroma in broad ligament, probably ovarian.

G 400. This section is taken from a recurrent tumor in the case just described. Microscopically it is a perfect reproduction of the original growth and indistinguishable from it.

During embryonic life portions of adrenal tissue frequently become detached and are scattered throughout the abdominal cavity. These adrenal rests have been found in the perirenal fat; on the surface of and in the substance of the kidney; in the liver; in the pancreas; in numerous other positions; and very frequently in the broad ligament. Although the presence of these rests is very common it is very unusual for them to take on a malignant development. The malignant tumors developing from adrenal rests in the broad ligament are extremely rare; and if, in our case, the development was from the hilum of the ovary, as we have at least negative evidence to show that it was, the case is unique.

The case most nearly approaching it is one

Fig. 9. No. 400. Same as Fig. 8 more highly magnified.

reported by Eastwood. But his tumor was purely a broad ligament mass, both ovaries being present and removed at the same time the tumor was removed. In our case the tube was intact, but the most painstaking search failed to reveal any ovarian tissue on the affected side.

ENDOTHELIOMA

No. 572. Mrs. L. Z., age 46, was admitted to the City Hospital, Apr. 27, 1907; has had eleven children, the last one eleven years ago; no miscarriages; last menstrual period eight years ago; no bleeding since.

She states that she was in good health up to eight weeks ago. Since then her abdomen has increased rapidly in size; there have been at irregular intervals severe cramping pains in the abdomen; no nausea or vomiting. The legs are greatly swollen. For the past five weeks respiration has been so embarrassed that she has been obliged to sit in a chair all the time; for the past two weeks there has been a severe cough. The bowels have been quite regular. Her strength has failed rapidly.

Examination. The patient is very feeble; the face is emaciated; expression anxious; respiration difficult; harrassing cough; costal breathing. The abdomen is much enlarged; the outline from the ribs to the pubes is symmetrical; the liver dullness is small and high; there is dullness over the lower part of the abdomen extending upward on the right side; the flanks are tympanitic; the legs are greatly swollen: the pelvis is filled with an irregular mass pushing downward. The urine contains albumin.

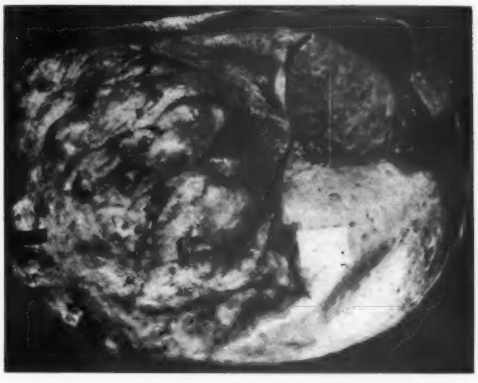

Fig. 10. No. 572. Hemangeo-endothelioma perivasculare of the ovary. This specimen has a solid and a cystic portion. In the solid portion is the ovarian tissue showing a small corpus luteum cyst. A large cyst 35 cm. in diameter springs from the ovarian tissue. The wall is thin and externally smooth; internally springing from the wall is a small cyst 4 cm. in diameter which is filled with dark fluid, and a nodule 2x2 cm. From the ovarian tissue numerous nodules project both on the free surface and into the cyst.

Fig. 11. No. 572. Low magnification showing cell masses around blood-vessels.

Apr. 30. There is some change in the apparent abdominal condition; the tympanitic area is now at the highest part of the abdomen when the patient is reclining; the dullness is in the dependent part. These percussion outlines point to ascites, while the outlines traced previously indicated the presence of a tumor.

Diagnosis. An ovarian tumor with ascites.

Operation. Apr. 30. The abdomen was opened through the left rectus muscle. A large quantity of clear yellowish ascitic fluid escaped. The parietal peritoneum was studded with nodules. A large tumor had developed from each ovary; the one from the right was pedunculated and was lying high in the upper right quadrant of the abdomen; the one from the left was partially cystic and partially solid; it extended from deep down in the pelvis nearly up to the lower border of the kidney; the whole tumor was densely adherent to pelvic and abdominal walls and to the

Fig. 12. No. 572. Higher magnification showing blood cells in lumen. Solid cell masses without any relation to blood-vessels are also shown.

intestine. The omentum was greatly thickened; the liver was covered with hard nodules.

The pedunculated, thick-walled cyst on the right side was removed.

May 1. Patient comfortable; slight nausea.

May 4. Abdomen filling up with fluid; no dyspnœa.

May 5. Quite a large quantity of fluid escaped from the incision.

May 8. The sutures removed; good union.

May 15. Patient is able to walk about the ward but is very weak.

May 21. Patient walked from the end of the ward to her bed, sat down and almost immediately died.

Histopathology. No. 572 A. This section was taken from a nodule projecting from the surface of the ovary into the attached cyst. The low-power view is that of masses of cells surrounding vessels cut longitudinally and transversely These cell masses are in the midst of larger areas of a pink-staining and structureless character. Under the high power the vessels are seen to be blood vessels with blood cells in the lumen and the endothelium of the intima intact. Immediately in contact with the adventitia are the tumor cells which form a complete investment to the transected vessels; when the vessel is cut longitudinally the investing cells can be seen to follow the bifurcations of the vessel. The cells in these areas average about 10 microns in diameter and consist of a nucleus which fills almost the entire cell, surrounded by a narrow rim of oxyphilic protoplasm. As the periphery of the cell groups is approached there is evidence of karyorrhexis and finally the cells no longer take up the nuclear stain, but show a faint outline staining with eosin wholly. Among these degenerated areas numerous red blood cells and an occasional leucocyte can be made out. This section shows plainly the origin from the perithelial tissue.

Fig. 13. No. 572. Low magnification showing well staining cell masses around vessels between which are unstained necrotic areas.

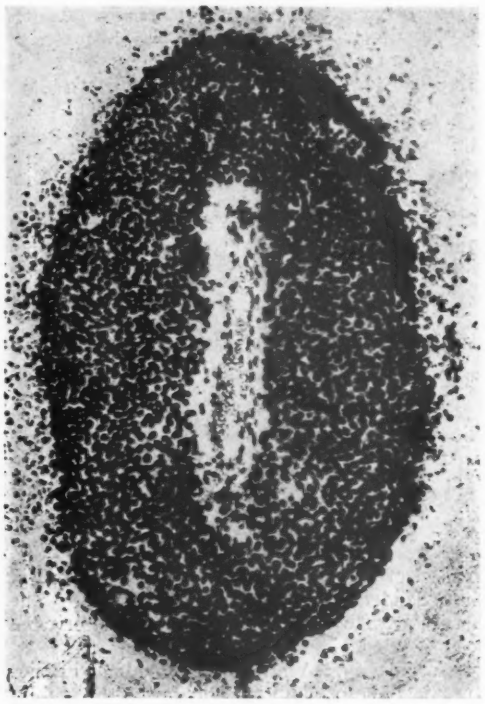

Fig. 14. No. 572. High magnification showing intima intact, the tumor cells arising in the adventitia. Surrounding the mantle of cells the tissue stains poorly with nuclear stains and shows the remnants of tumor cells mixed with extravisated blood.

572 C. The polymorphous character of the cells and the density of their arrangement when proliferation is well advanced is well shown. The tendency of the cells to arrange themselves around lumen in which frequently a hyaline mass can be seen is here well shown and is a characteristic feature of tumors of endothelial origin. Other fields resemble carcinoma inasmuch as there are well-defined alveoli and the cells in structure closely resemble epithelial cells. In other places the structure suggests that of large round cell sarcoma. However the origin is so unmistakenly shown in section G 572 A that there can be no hesitancy in classifying it as a hæmangioendothelioma perivasculare.

An endothelioma is a tumor originating in endothelium.

"They simulate morphologically the atypical epithelial tumors and histogenetically the atypical connective tissue tumors.

"The ability of endothelial cells to undergo metamorphosis simulating the epithelial type at one place and the connective tissue type at another has led to endless confusion in classification. Hence we have 'alveolar sarcoma,' 'endothelial sarcoma,' 'angiosarcoma,' etc. Unfortunately we can not always see the earliest steps in the origin and growth of these tumors but must depend on interpreting these processes by histologic examinations.

"Embryologically these tumors belong strictly to the connective tissue group, but endothelium stands midway between epithelial and connective tissue cells. Thus flat endothelium may give rise to cylindrical cells resembling epithelium, or may form spindle cells, or appear as fibroblastic elements. They differ from ordinary connective tissue cells by their ability to secrete mucin and hyalin. Like connective tissue cells they produce granulation tissue, and epitheloid and giant cells. They may form tubules lined with a layer of cylindrical cells like epithelium." (Zeit.)

According to whether the endothelial proliferation originates from lymph vessels and spaces or from blood vessels and whether the inner endothelial lining or the outer proliferates we have:

1. Lymphangioendothelioma intravasculare.
2. Hæmangioendothelioma perivasculare.
3. Hæmangioendothelioma intravasculare.

The origin of our tumor is strikingly apparent; the proliferation is easily seen to begin in the so-called perithelium of the blood vessels and to ensheathe them in a mantle of cells which follows the bifurcations of the vessels. In areas removed from the seats of origin the growth assumes features entirely different, strongly suggestive of carcinoma in places, but, in such places, the cells arrange themselves so as to produce well-defined lumina, giving a characteristic sieve-like appearance. If such areas alone were examined it would be impossible to determine with certainty the origin of the growth.

Clinically, endotheliomata are malignant and are exceptionally prone to recur at the seat of operation much earlier than either sarcoma or carcinoma and the growth is usually very rapid. They may occur at any age.

The type of tumor here illustrated is that best classified as hæmangioendothelioma perivasculare. In this variety, the growth begins in the so-called perithelium, i. e., the endothelium outside the adventitia, and the proliferated cells, either round or oat-shaped, are radially disposed around the blood vessels.

CARCINOMA

No. 466. Mrs. A. F., age 56, admitted to the City Hospital, Oct. 25, 1906; has had one child

Fig. 15. No. 572. This section shows the tendency for the cells to arrange themselves around a lumen giving a sieve-like appearance to the tumor mass.

twenty-eight years ago; no miscarriages; menses ceased seven years ago, before that normal.

She has been unwell for the past year; eight months ago, first noticed a swelling of the abdomen which has gradually increased; has slight nausea; marked constipation.

Examination. The abdomen is enlarged to the size of a six months' pregnancy. By palpation and percussion a rounded cystic tumor can be easily made out occupying the lower half of the abdominal cavity; the uterus is pushed downward and forward by a mass which is more resistant on the left side. The general condition of the patient is apparently good.

Diagnosis. Ovarian cyst.

Operation. Oct. 27. The abdomen was opened through the left rectus muscle; the cyst was tapped and about one and a half quarts of fluid were drawn off; the opening was closed by a clamp, a few adhesions about the lower part of the growth were broken up, the pedicle tied off and the tumor removed. The tumor originated in the left ovary and there was no evidence of extension except a little thickening of the broad ligament.

The convalescence was uneventful; discharged Nov. 12.

July, 1908, patient reported in good health.

Histopathology; No. 466-B. The specimen shows numerous loculi of varying sizes which vary widely in their contents. The larger ones are beset with papillæ and contain a mucoid material. The connective tissue element of the wall is typically that of the ovarian cortex, rich in spindle-shaped cells. In other loculi, there is unmistakable evidence of active proliferation of the epithelial covering of the papillæ as shown by the reduplication and numerous free epithelial cells which almost, or completely, fill the loculi. These cells are large with a relatively small amount of cytoplasm. In some loculi, the papillæ are absent and the compartments are completely filled with solid epithelial masses.

There is a tendency to myxomatous degeneration of the stroma in certain parts of the section.

Diagnosis. Carcinoma of ovary originating in papillocystoma.

No. 3087. Mrs. K. N., age 48, admitted to the City Hospital, Sept. 18, 1903, has had five children; no miscarriages; the last child was born Aug. 12, 1898. Last menses Oct. '02; before that, irregular for five years

Her present complaints date back only four or five weeks, since when she has had a moderate amount of pain in her right leg and some pain in the bladder and frequent micturition. The bowels have been regular. She first observed a mass in her abdomen one week ago.

Examination. A firm, irregular tumor extending above the pelvic brim half way to the umbilicus could be easily palpated; by the vagina, a hard,

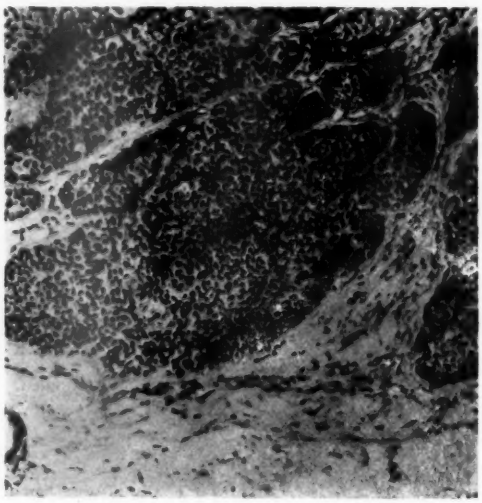

Fig. 16. No. 572. This section illustrates the carcinoma-like arrangement frequently seen in these tumors.

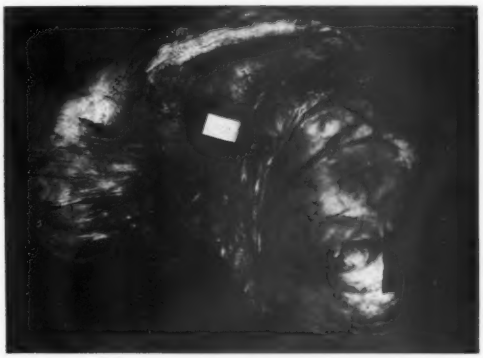

Fig. 17. No. 466. The tumor consists of an evacuated cyst 20 cm. in diameter, with smooth walls, and a solid portion 3.5 cm. thick. Stretched over the top of the mass is the tube. The solid portion on section is soft but not disorganized.

slightly movable mass could be felt filling the pelvis and projecting into the cul-de-sac.

From the pressure symptoms and their recent appearance, and from the firm, palpable growth, we supposed that we had a fibroid to deal with.

Sept. 19. Operation. When the abdomen was opened the mass was found to be made up of two growths, one from each ovary, which filled the pelvis, were adherent to each other and to the entire pelvic wall. The adhesions were broken up without difficulty and the whole mass and the adherent uterus were removed. A number of large metastatic growths were observed in the mesentery.

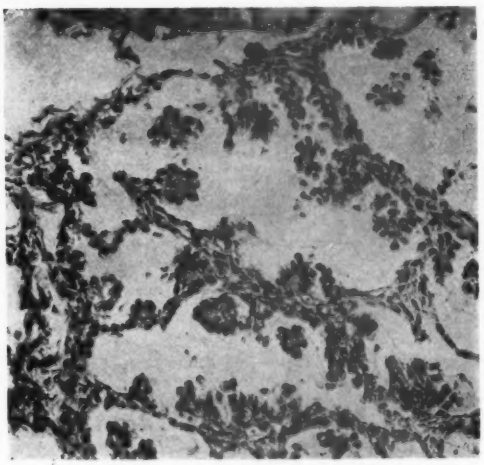

Fig. 18. No. 466. The section shows epithelial proliferation and beginning disappearance of the stroma. In some loculi the epithelial cells fill up the entire space.

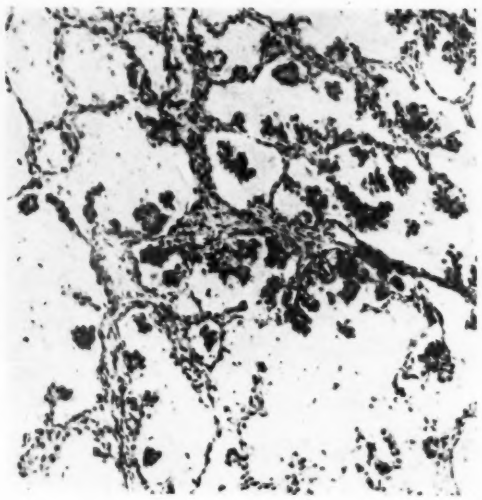

Fig. 19. No. 466. Very similar to Fig. 18.

The convalescence was uneventful; she went to her home in a confortable condition Oct. 12.

This patient remained in apparently good health for several months and then succumbed to the progress of the disease.

Histopathology. No. 3087. There are numerous large loculi, some as large as 1 mm. containing a colloid material imbedded in which are desquamated epithelial cells. These loculi are lined with epithelial cells, six or eight layers thick. The outer layers are columnar in shape, while those nearest the cavity are spheroidal.

The major part of the section consists of solid masses of epithelial cells lying in a rather poorly developed connective tissue stroma.

The cells composing the masses are chiefly spheroidal and there are no blood vessels or stroma between them. The stroma is rather poorly supplied with blood vessels.

There are many loculi varying from 75 to 250 microns which consist of a fibrous wall lined on the inner side with proliferating epithelial cells which as yet do not completely fill the cavity, leaving a clear lumen.

Diagnosis. Carcinoma of the ovary, probably metastatic.

No. 548. Miss M. J., age 51, admitted to the City Hospital Mar. 4, 1907; no pregnancies; menses regular, moderate in amount and painless up to seven years ago, when they ceased.

No. 3087. The specimen consists of the uterus and tubes and two masses one involving each ovary which unite below to form one large mass. There is a cyst 4 cm. in diameter, filled with colloid, at one side.

Fig. 20. No. 3087. Adeno-carcinoma of both ovaries. This specimen consists of the body of the uterus, which was amputated at the internal os, and the two large growths from the ovaries which are adherent to the uterus and to each other posterior to the uterus. The growths are solid.

Fig. 21. No. 3087. Low magnification showing nests of epithelial cells in a dense fibrous stroma. No cysts.

One year ago she first noticed an enlargement of the abdomen; since then it has gradually increased in size. In Nov., 1906, there was some bleeding from the uterus; this flow has continued to the present time. It has never been profuse and consists of clear, bright blood. On Feb. 6, she went to bed on account of abdominal pain and has remained there since. She has been able to take very little food for several weeks.

Examination. The patient is emaciated and very weak; the abdomen is smooth and tense and enlarged to the size of a full-term pregnancy; there is tenderness on palpation of the tumor; this tenderness is most marked over the lower left quadrant: by the vagina a mass can be felt on both sides of the uterus and filling the cul-de-sac. The urine shows a small amount of albumin.

Diagnosis. Ovarian cyst.

Operation. Mar. 5. The incision was made a little to one side of the median line. The cyst wall was very friable and was broken through allowing a large quantity of dark thick fluid containing pieces of necrotic tissue to escape. The part of the tumor deepest in the pelvis was solid; the remainder had the appearance of a necrotic multilocular ovarian cyst. It had developed from the right ovary. The extreme friability of the cyst walls and the adhesions to the intestines and to the pelvis made the tumor very difficult to remove. The left ovary was enlarged to a nodular tumor the size of the first; it was removed. Very little blood was lost; two large cigarette drains were put in.

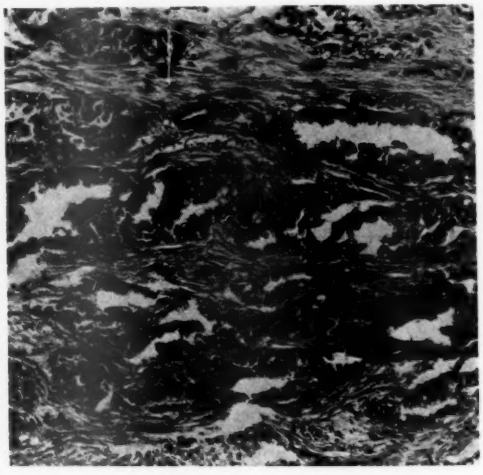

Fig. 22. No. 3087. Higher magnification of the same section shown in Fig. 21.

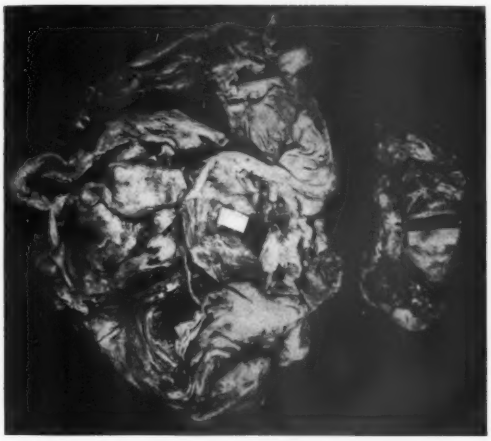

Fig. 23. No. 548. This specimen consists of two portions. The one from the right ovarian region consists of an irregular mass with the free fimbriated end of the tube attached. There is a cyst wall and a solid portion; the latter is made up of nodules averaging 3 cm. in diameter. The cyst wall being very friable was much broken up in the process of removal. The left ovary is a mass 7 x 5 cm. with a small adeno-cystoma at one pole. A normal tube with free fimbria is attached.

Fig. 24. No. 548. Low magnification showing proliferating epithelial cells lining loculi. In some places it is slight in others it has gone so far as to form solid masses.

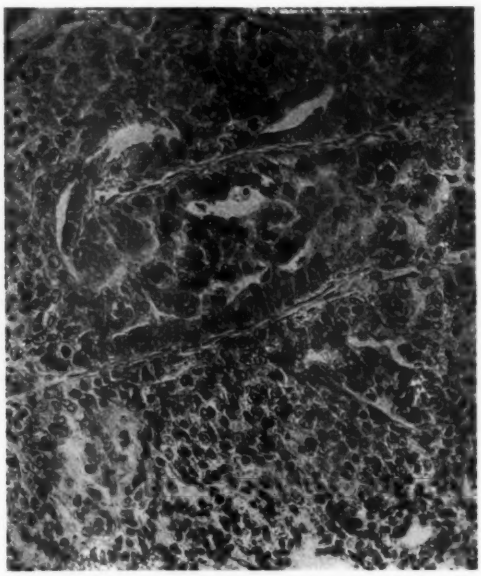

Fig. 25. No. 548. The same section shown in number twenty-four more highly magnified.

The convalescence for a few days was stormy; the trouble was due to inefficient drainage

Histopathology. No. 548 C. Practically the entire section is extremely cellular and under the low magnification appears to consist almost en-

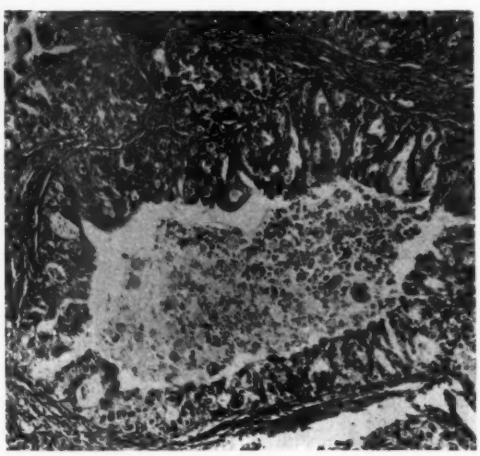

Fig. 26. No. 548. Low magnification of the denser portion of the growth.

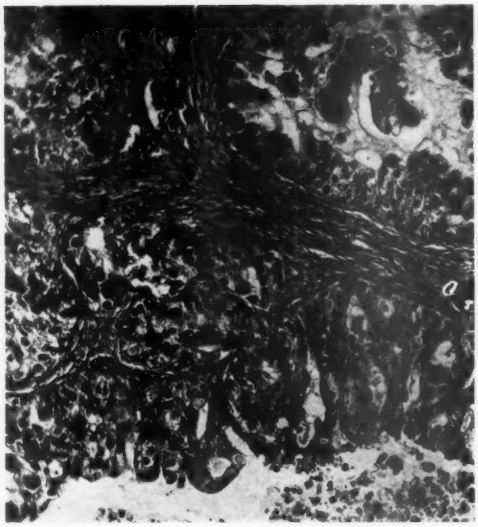

Fig. 27. No. 548. High magnification of the same section shown in number twenty-six, showing the breaking down of the wall between adjoining loculi and the solid masses of carcinoma.

tirely of closely packed epithelial cells. With higher magnification alveoli, with quite thin walls, of varying sizes, completely filled with closely packed epithelial cells can be made out. There is every evidence of rapid growth as shown by bizarre forms of nuclei. Nucleoli are especially prominent in the majority of the cells. At the periphery of the section, several loculi are seen where the epithlial lining bends to arrange itself after the type of a tubular gland. These tubules are cut in long or cross section. The cavities of these loculi contain disintegrated epithelium and a colloid substance.

Diagnosis. Carcinoma of ovary originating in adenocystoma.

No. 535. Mrs. I. G. age 46, admitted to the City Hospital, Feb. 1, 1907; has had five children, the last one ten years ago; menses ceased eighteen months ago.

Six months ago she began to have pains in the back and the abdomen commenced to enlarge. Since then she has not felt well.

Examination. General condition good; the abdomen is enlarged to the size of a full-term pregnancy; outline irregular; the outlines of a large cystic tumor easily made out; the uterus was pushed down by the mass.

Diagnosis. Ovarian cyst.

Operation. The incision was made through the right rectus muscle; the abdominal wall was very thin; a few adhesions were broken up, the fluid drawn off and the cyst wall and the solid portion of the tumor brought outside; the pedicle was clamped and tied and the mass removed.

The convalescence was uneventful; discharged Feb. 22.

July 24, 1908. Patient reports by mail that she has been perfectly well since operation

Histopathology. No. 535. The section is from the solid tissue of the tumor including the capsule.

The capsule consists of dense fibrous tissue mixed with smooth muscle, and sends prolongations of varying size into the tumor mass forming loculi in which the tumor cells lie. At one point beneath the capsule some typical cortical ovarian stroma is seen. The larger branches of the framework contain numerous blood and a few lymph vessels. The majority of the loculi are competely filled with epithelial cells which in numerous places have broken through the intervening septa transforming adjoining smaller loculi into one large one. The epithelial cells which are nearest the wall of the loculi show a tendency to a gland-like arrangement. They are columnar in shape and from them the proliferating cells extend into the loculus and fill it. The centrally disposed cells are chiefly spherical or oval in shape and between them neither blood nor lymph vessels can be demonstrated. Neither is there any intercellular stroma except the remains of broken-down septa where adjacent loculi have coalesced.

Fig. 28. No. 535. Carcinoma of the ovary originating in an adeno-cystoma. Large multilocular cyst with smooth walls. The tube is stretched over the cyst. The ovarian tissue in which several corpora lutea can be seen is embedded in a mass of newly formed tissue. In this tissue is an adeno-cystoma 5 cm. in diameter.

The growth is carcinoma of the ovary, evidently originating in an adenocystoma.

Ovarian carcinomata are either primary or metastatic. In both forms there is a marked absence of symptoms other than those caused by the intraabdominal tension brought about by the tumor and the commonly accompanying ascites. There is frequently nothing to suggest malignancy. They are most common after forty but are met with at all ages.

In cases of primary carcinoma evidence of previous pathological but benign changes, has been found in most specimens. In the early stages, the capsule is firm and smooth; later when the tissue proliferates in it and penetrates it, it becomes friable and rough. The carcinomatous growth is usually found in the thickened portion of the cyst walls. The epithelial cells are derived from the lining of the follicles and from the cells derived from the germinal epithelium. Metastatic growths from primary carcinoma of the ovary depend largely upon the integrity of the capsule of the tumor. Recurrence is probable and the mortality is high.

In metastatic cancer of the ovaries the primary lesion may be in the stomach, the intestines, the gall-bladder or ducts, or in fact any of the abdominal organs. Bland-Sutton has repeatedly called attention to these metastatic growths in the ovary. He states that "Schlagenhaufer collected and analyzed 79 cases of malignant disease of the ovaries associated with primary cancer of the abdominal viscera; of these, in 61 instances the primary focus was in the stomach, 10 in the bowels, 7 in connection with the gall-bladder and ducts and one in the adrenal gland. The majority of the patients had been submitted to operation, and the ovarian tumors were removed under the impression that they were the primary disease, the cancer in connection with the intestinal tract being in most instances overlooked until after the fatal termination of the case."

Schlagenhaufer's conclusion from his researches is that the majority of bilateral solid tumors of the ovaries are carcinomata and metastatic. The histologic features of these ovarian tumors vary with the nature and situation of the primary growth. The practical deduction is that in all cases of bilateral solid tumors of the ovary, the surgeon should make a careful examination of the abdominal organs, especially the stomach.

The explanation of the process of metastasis from the upper abdomen to the ovaries is made in this way; the cancer starting in the mucous membrane gradually penetrates the submucous and muscular coats and infiltrates the peritoneal

Fig. 29. No. 535. Low magnification showing the proliferation of the epithelial lining of the cysts.

covering. The cancer cells escape from this surface into the peritoneal cavity, are distributed by the fluid and the intestinal movements and gradually fall into the pelvis, and come in contact with the ovaries.

In a very recent article, Bland-Sutton says: "It is easy to understand, now the matter is explained, why the true pathological conditions involving bilateral carcinoma of the ovary have been misunderstood. Few of us can have realized that metastatic cancerous masses could attain such large proportions in so small an organ as the ovary; there must be something favorable in this gland to allow cancer cells to engraft themselves and form such a large tumor. That the primary growth in the gastrointestinal tract should be overlooked is not altogether a matter of surprise for they are often small and inconspicuous in the colon, and as a rule in an ordinary ovariotomy the stomach is not seen. Even in patients in whom there is dissemination throughout the abdomen, this is usually regarded as arising from the ovarian tumor, instead of it being realized that the masses in the ovaries, like the nodules in the intestines and viscera, are secondary deposits.

"It should also be remembered that the slow and insidious manner in which carcinoma grows in the colon, and especially in the gall-bladder, tends to make diagnosis difficult and uncertain."

Two of the cases, No. 466 and No. 535, were

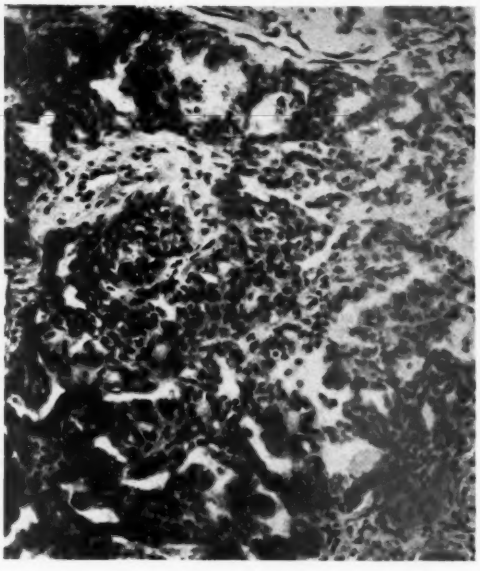

Fig. 30. No. 535. Same as number twenty-nine more highly magnified.

primary cancer of the ovary. They were unilateral; the capsule of the tumor in both cases was unbroken. And up to the present time there has been no recurrence. No. 548 was probably a primary cancer beginning in an adenocystoma of the right ovary, but the process had involved so many other organs that it was impossible to locate the origin with precision. No. 3087 had all the ear-marks of a metastatic growth; it was solid, bilateral, did not show evidence of a preceding benign pathological process, and was associated with numerous metastatic foci throughout the abdomen. The primary focus was not located.

SARCOMA.

No. 362. M. M. age 45, admitted to the City Hospital June 5, 1906; last menses Dec., 1905; for several months before that the flow had been scanty.

In Jan., 1906, she first noticed an enlargement of the abdomen; it has gradually increased in size up to the present time; there has been some disturbance of the bladder; the appetite poor; nausea has been especially marked during the past few months; the bowels are constipated. This combination of symptoms led her physician to suspect that she was pregnant and she was sent to the obstetrical department of the hospital.

Examination. General condition poor; anæmic; pulse about 100 per minute and weak; breathing shallow; mental condition dull. The abdomen is enlarged to about the size of a full term pregnancy; the tumor is smooth, tense and no solid portion can be felt; the whole abdomen is dull on percussion except for a short space below the ribs and in the flanks. The uterus is not enlarged and is pushed down by a mass that fills the pelvis. The breasts show no signs of pregnancy. Urine normal.

Diagnosis. Ovarian cyst.

Operation. June 15. The tumor was a cyst of the right ovary; about one gallon of thick, dark fluid was drawn from it; its walls were nodular and adherent to everything coming in contact with it; the peritoneum was thickened and nodular; the omentum was thickened and contained many nodules; large masses of thick cheesy exudate and much free pus was found in the peritoneal cavity; drainage.

June 16. Died at 2 P. M.

No. 362. This section was made from a nodule on the wall of the larger of the ovarian cysts. The tissue is composed chiefly of spindle cells scattered among which are occasional large and small round cells. The intercellular substance is scant and has no regular arrangement. The blood vessels with thin walls are among the cells. At

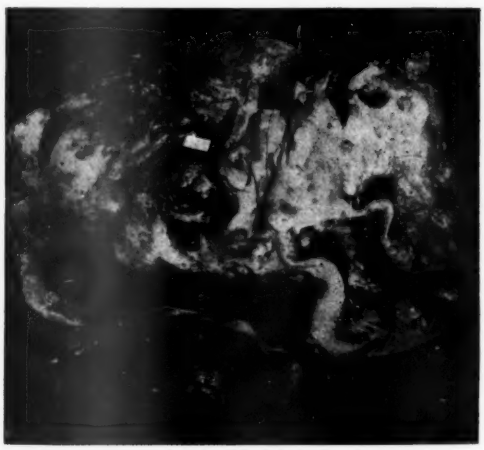

Fig. 31. No. 362. Mixed cell sarcoma with infection in an adeno-cystoma of the ovary.

The specimen consists of two distinct cysts, one 50 cm., and the other 30 cm. in circumference. The walls of both cysts are studded with nodules. On the walls of the larger cyst is the remaining ovarian tissue which contains an adeno-cystoma 10 cm. in diameter.

Fig. 32. No. 362. Low magnification showing spindle cells and round cells. The lighter areas are infiltrated with leucocytes and contain broken down tissue.

Fig. 33. No. 362. Higher magnification of the same section shown in Fig. 32.

several points there has been hæmorrhage into the tissue and blood pigment is seen in the leukocytes and free in the tissues. An acute infective process is present in the growth, as there are areas of necrosis surrounded by a typical leukocytic zone.

The tumor is a mixed cell sarcoma with infection.

No. 571. M. D. colored, age 54; admitted to the City Hospital Apr. 23, 1907; never pregnant; menses normal up to six years ago, when they ceased entirely.

She has had no discomfort until the past few weeks; did not notice the enlargement of her abdomen until two weeks ago, though her friends spoke of it a couple of months ago. She first had pains in the legs which afterward extended to the abdomen. Has nausea but has not vomited; shortness of breath; thinks she has lost fifty pounds in the past three years.

Examination. The abdomen shows an enlargement extending from the ribs to the pubes; it is rounded and regular except at the umbilicus where a hernia as large as a hen egg projects. The right side of the abdomen is the more prominent. On percussion, a dull note is obtained from the umbilicus down; the flanks and the area above the umbilicus are tympanitic. The pelvis is filled with a mass pushing down from above. No swelling of the feet or legs; the urine contains large quantities of phosphates, but no albumin or sugar.

Diagnosis. Ovarian cyst.

Operation. Apr. 30. The abdomen was opened through the left rectus muscle; the cyst was found firmly adherent to the parietal peritoneum; the sac was opened and a large quantity of thin, dark fluid was removed; the cyst wall was nodular and very adherent to the peritoneum and the intestines. In separating these adhesions the friable cyst wall was torn into many pieces and at one point a large hole was torn in the small intestine. This opening was immediately closed by sutures. The tumor had developed from the right ovary, the pedicle of which was tied off. The peritoneum was studded with small nodules; the omentum was thickened and nodular. There was considerable oozing from the broken adhesions but no great amount of blood was lost. Four large gauze drains were used.

May 1. Patient bright and with no complaints.

May 20. The wound not entirely closed; patient gaining in strength.

May 28. General condition quite good; patient able to walk about.

June 2. For the past two days there has been a slight rise of temperature and pulse; the patient has felt badly and has remained in bed; to-day at 1 P. M. she complained of pain in the right side; almost immediately she became unconscious and within a few minutes died.

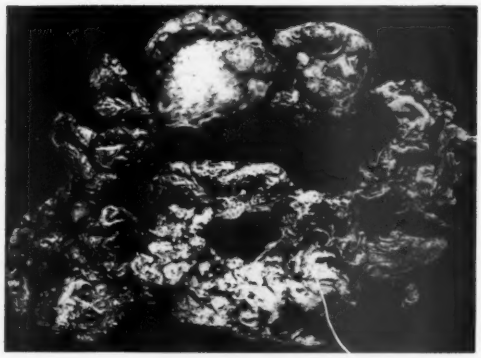

Fig. 34. No. 571. Spindle cell sarcoma of the ovary. This tumor was so friable that in removing it was broken up into a shapeless mass of tissue in which no relations to any organ can be made out. There is no sign of tubes or ovaries.

No. 571 A. The structure of the growth is typically one of spindle cell sarcoma, with laminæ in long and trans-section. The blood vessels are thin walled and immediately in contact with the tumor cells. Side by side with well-staining tissue, as shown in the illustration, are areas of degeneration consisting of tumor cells, blood corpuscles and detritus.

The practical clinical conclusions to be drawn from the study of malignant tumors of the ovaries are:

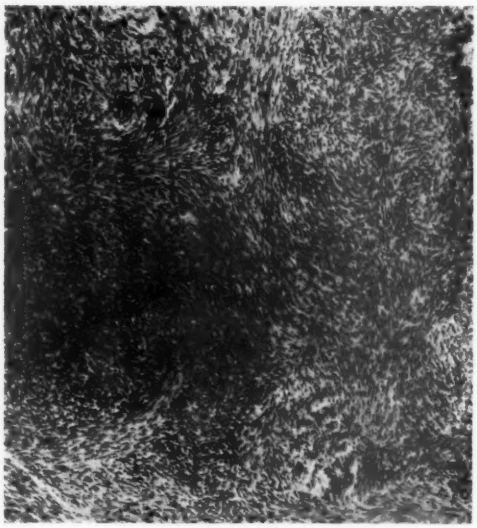

Fig. 35. No. 571. Low magnification showing bundles of spindle cells in variously disposed lamina.

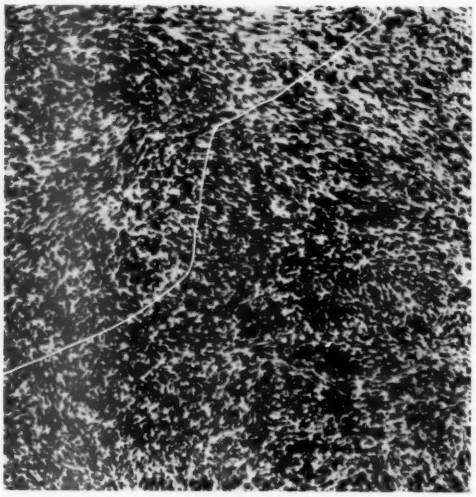

Fig. 36. No. 571. Higher magnification of the same section shown in Fig. 35.

First. All ovarian tumors should be removed early, because primary carcinoma of the ovary usually begins in a benign growth and an early operation removes a prolific source of malignancy. Even where the malignant process has made considerable progress there is an excellent opportunity to effect a complete cure.

Second. Ovarian tumors should be removed through an abdominal incision long enough to allow the tumor to escape without breaking the cyst wall. We must come back to what Washington Atlee called the "major incision"—the incision that is most strenuously advocated today by the sanest of the clinician-pathologists, J. Bland-Sutton.

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THE SURGICAL TREATMENT OF TUBERCULOUS ADENITIS, WITH SPECIAL REFERENCE TO THE CERVICAL LYMPHATICS

By J. A. ATTRIDGE, M. D., Detroit, Michigan

There has been a good deal of discussion as to the most satisfactory manner of treating tuberculous adenitis, and it has been my aim to bring before you some data which to me seem quite conclusive.

The most important literature comes from European and American clinics, where the cases treated have been kept under observation for long periods of time, thus enabling us to know the ultimate outcome of these patients. To me, it seems as if this is the only satisfactory way of definitely settling this very important point.

The tendency to infect internal organs is best shown by the records of cases treated non-surgically and in those where surgical procedures stopped short of complete removal of the infected nodes and other infected tissues.

Demme, reported by Dowd, reviews the first twenty years' work in the Children's Hospital in Berne, in which six hundred and ninety-two patients with lymph-node tuberculosis were treated by constitutional measures, not by surgery, with the following results:

	Cases	Per cent
Developed tuberculosis of the lungs	145	21.0
Of the intestine	24	
Of the pia mater	25	
Of the kidney	6	
Of the epididymis	2	
Total	57	8.2

Or a total of 29.2 per cent.

This does not give the number of bone, nor of the late lymph-node infections. The period of observation was in some cases short, and therefore, the extent of involvement of internal organs was not determined.

"Statistics from large German clinics show that 25 per cent of cases not treated surgically, subsequently develop pulmonary tuberculosis. About 10 per cent tuberculosis meningitis and a smaller per cent of bones and joints."

Van Noorden gives 28 per cent of tuberculosis in a series of one hundred and forty-nine patients, that were observed from three to sixteen years after operation.

In a series of one hundred and sixty cases reported by Blos, which were observed for from three to twelve years after operation, statistics show 26 per cent of lung tuberculosis and 14 per cent tuberculosis in other organs.

Fisher has tabulated from the literature twelve hundred and seventy-three cases that were observed from one to sixteen years after operation, as follows: Cured, 57.65 per cent; local recurrence, 21.84 per cent; died almost entirely from tuberculosis, 13.51 per cent.

Karwoski reports a series of one hundred and twenty-eight cases in children that were operated upon and kept under observation for a long period of time with only three deaths from tuberculosis trouble, or 2.5 per cent.

In people of all ages, Bowilly gives 10 per cent mortality; Bruhn 10 per cent; Grunfeld 10 per cent; Scheyer 11.5 per cent; Von Burgman gives 29 per cent of cured in people who had the gland infection; as children 43 per cent improved; 22 per cent somewhat improved; 6 per cent not improved. Vogleman 52 per cent cured; 29 per cent improved; 19 per cent not improved and reports two hundred and fifty cases operated upon without untoward results. The operative treatment gave 70 per cent of cures; general treatment only 24 per cent. He cites two cases where operation was refused and in one case after six months, and in the other, after two years, miliary tuberculosis caused death. Karwoski makes the statement that the only reason for not removing the tuberculosis nodes, are extreme weakness, or extensive tuberculosis of internal organs, where favorable results could not be expected.

This report would seem incomplete if it failed to show the results in a series of tabulated cases by Wohlgemuth, showing the relative value of different methods of treatment:

	Per cent	Per cent	Per cent
No operation	24	37	39
Incision and curetting	63	27.7	8.3
Extirpation	70.5	22.8	6.4

The literature shows that large numbers of cases have been treated by general treatment, together with serums and local measures, to determine their value. In one series (Halsteads) internal, surgical, and open air treatment, combined under the most favorable circumstances, have been practised.

The more a person studies the subject, taking into consideration the glands infected, the mode of infection, the tendency to metastasis, the result from different lines of treatment — the more con-

vinced is he, that thorough removal of the infected tissues offers the quickest and only hopeful outlook for a large percentage of these patients. In support of this contention, I again quote Wohlgemuth's series of 75 per cent cures from complete removal of node; 63 per cent from incision and curettage, and 24 per cent from general treatment.

Dowd's one hundred complete operations with one case of pulmonary tuberculosis and three cases of bone tuberculosis following. Freedom from recurrence in about 75 per cent and ultimate recovery in about 90 per cent in most favorable cases; in less favorable cases, freedom from recurrence in 50.55 per cent; ultimate cure in 70.75 per cent of cases.

Jordan, from an experience of four hundred cases treated by operations of various kinds, strongly urges complete excision of the nodes.

Keen removes all the nodes he can find, whether enlarged or not, believing that if not enlarged, they may be infected, and will enlarge.

Gibson believes the outlook for lymph-node tuberculosis, if prompt removal is instituted, is more hopeful than is operation in bone tuberculosis. He also believes that lymph-node infection is nearly always primary and when they are not removed that lung, intestinal, and cerebral involvement is frequent.

Mayo's two hundred and thirty-five operations for primary tuberculosis, and fifteen for secondary tuberculosis in the past four years, seem to confirm this opinion.

Burrell believes that where it is possible the tuberculosis glands should be excised if they threaten to invade vital structures.

Sutcliff (British Medical Journal) from personal experience and observation of several hundred cases, has seen several cases of tuberculous meningitis, succeeding tuberculous lymph glands. He has only seen one case follow operation on the gland, and in this case the patient did not stand the operation well and an incomplete operation was the result. He believes that when the operation is decided upon, the glands should, in all cases, be removed with surrounding infected tissues, as meningitis and bone infections are less liable to follow complete, than partial operations.

Dowd believes that every case with tuberculous cervical lymph nodes should be operated upon, unless there is good reason to believe the operation would not be endured.

C. H. Mayo from an experience of five hundred operations, believes that tuberculous glands are best treated by their removal.

In acute tuberculous adenitis, the diagnosis from Hodgkin's disease, simple hyperplasia, and syphilis is difficult. In the more chronic conditions there is a greater frequency of tuberculous adenitis in children, and the primary location is in the deep group of glands under the sterno-mastoid muscle, while in Hodgkin's disease the anterior and posterior triangles are more frequently primarily involved and the disease is found in young adults.

Tuberculous glands are usually larger above, where infection begins, and gradually smaller as you follow them toward the thoracic duct, with a greater tendency to suppuration. Tuberculous glands are more frequently confined to one group of lymph nodes.

In syphilis there are other manifestations of the disease and where the gland has a tendency to break down there is usually a boggy or œdematous condition of the adjacent tissues, which I have not observed in tuberculous glands.

At the present time many of the glands that were considered simple hyperplasia are now looked upon as tuberculous, consequently leaving a certain number of cases in which a positive diagnosis must depend on microscopical examination of removed tissues. Sarcoma and carcinoma must be kept in mind. The nodes first involved depend upon the source of infection.

It is generally agreed that the cervical glands are infected primarily from the tonsils, either faucial or pharyngeal, decayed teeth, gums, scalp or some lymphoid tissue in the pharynx, unless some external lesion is apparent. That infection through the blood stream is not frequent is explained; since examination of the chyle stream has shown that where the bacillus was absorbed by the intestines, it was taken up by the lacteals and reached the blood stream by way of the thoracic duct.

That infection usually takes place through the nose and throat,— 86 per cent in Dowd's cases — is easily understood by referring to the relation of the cervical lymphatics to the above-mentioned infection atrium. To substantiate the correctness of this view, Wood, in 1905–1906 conducted a series of experiments, by injecting the tonsil, showing the direction of the lymph current.

Contrary to the views held by some anatomists, that the tonsils first emptied into some of the superficial set of glands, and they in turn into the deep chain, he found the faucial tonsils emptied directly into the internal deep gland or tonsillar gland, while the pharyngeal tonsils emptied into the external divison of the deep chain, posterior to the sterno-cleido-mastoid muscle.

The superficial plexuses of lymphatic glands,

namely: Occipital, mastoid, parotid, submaxillary, and submental receive their lymph current as indicated in Plate 1, and empty in the deep jugular plexus (Plate 2), thus reaching the venous circulation by one of the points indicated in Plate 2. When either of these plexuses becomes infected you can trace the infection by the afferent supply channels, unless the deep glands are first involved, when secondary infection of glands would indicate choking of the natural lymph channels, and thus backing up the infected stream in an effort to establish collateral circulation (Plate 2).

While extirpation of the glands gives the best results, there is still a field for incision, curettage and general treatment, which includes drugs, sanitation and out-door life.

I believe that much judgment is necessary in deciding when and how to treat an individual case. A radical operation in acute tuberculous infections before the disease becomes well localized, and when the anti-tuberculins and other auto-bodies are exhausted by reason of a severe infection, is not indicated; such cases can be best treated tentatively by rest, hygiene, reconstructives, out-door life, and X-rays in some cases, until the acute symptoms subside, when a thorough removal of the glands should be made. Should a gland suppurate during the interval, it should be incised, curetted, and the edges of the incision and cavity treated by a stimulant, as iodine, or other stimulating antiseptic, which temporarily arrests the action of bacteria and promotes sufficient serous flow to block up the avenues to infection. Avoid prolonged drainage. The incision will close in a short time, then a later complete removal of glands is indicated.

In the more chronic cases where it is difficult to distinguish between tuberculosis and simple hyperplasia or Hodgkin's disease, an examination of the portals of entry, usually the nose and throat, should be made. If enlarged tonsils, adenoids, or other infection atrium are present, they should be immediately removed and examined microscopically for the bacillus. If found, this not only gives us a definite knowledge as to the form of infection, but prevents further infection and is a necessary part of the treatment in every case.

In the chronic cases with sinus formation, mixed infection is probable. These cases should be thoroughly disinfected, curetted, and allowed to heal, when the glands should be removed.

Alexander Hugh Ferguson's only mortality occurred in a case with mixed infection. The difficulty attending the removal of these glands must appeal to all who consider the complex arrangements of structures, some of them vital, immediately surrounding, and in some cases, adherent to, parts to be removed. Particularly is this true, when the infection has passed outside of the gland, to surrounding tissues, and in cases where there have been discharging sinuses.

The incision should be as little disfiguring as possible, consistent with opening up a large, otherwise inaccessible field. From personal experience and observation, I am convinced that previous to cutting down on the enlarged gland, we cannot always determine the extent of involvement. What appears as a single enlarged gland in the sub-parotid region, after incision, will reveal a number of enlarged glands in the deep chain, beneath the upper portion of the sterno-mastoid.

Dr. C. H. Mayo removes tuberculous glands through an incision (Plate 3), which begins one inch below and posterior to the tip of the mastoid bone. This extends down the border of the trapezius, half-way to the clavicle. It then follows the natural skin creases, or lines curving forward to the anterior sterno-clavicular region, ending between the divisions of the sterno-mastoid muscle. The platysma and external jugular are cut and tied and elevated with the skin. The superficial ascending cervical nerves (Plate 1), are divided, but like other purely sensory nerves they soon unite and function is restored.

The spinal accessory nerve (Plate 1), is freed, commencing above under the sterno-mastoid and followed to the border of the trapezius. Injury to this nerve may result in drop shoulder, atrophy, and spinal curvature in the young; a number of such cases are reported. If the nerve is severed, the ends should be found and sutured. The sterno-mastoid muscle is retracted forward and dissection commenced at the lower angle of the wound. The intimate relation of the nodes to the internal jugular (Plate 2), prompted Fenger to tie this vessel as a preliminary step in the operation. The dangers attending injury to this vein are hæmorrhage, air embolism, and aspiration infection, in cases where suppuration has taken place. This complication can be avoided by placing pressure on the vein, low down, where the vessel passes under the omohyoid muscle. The pressure causes the vein to fill, and, if cut, it will bleed and not aspirate air (Murphy, Mayo). The vein has been tied and removed with gland in a few instances without untoward symptoms, but three deaths from this practise (Lender, Krummer, Rohsback) show that it is not without danger. Having reached the afferent lymph vessel below the last enlarged grand, a clamp should be used to prevent crowding of infection into it, during subsequent manipulation of glands.

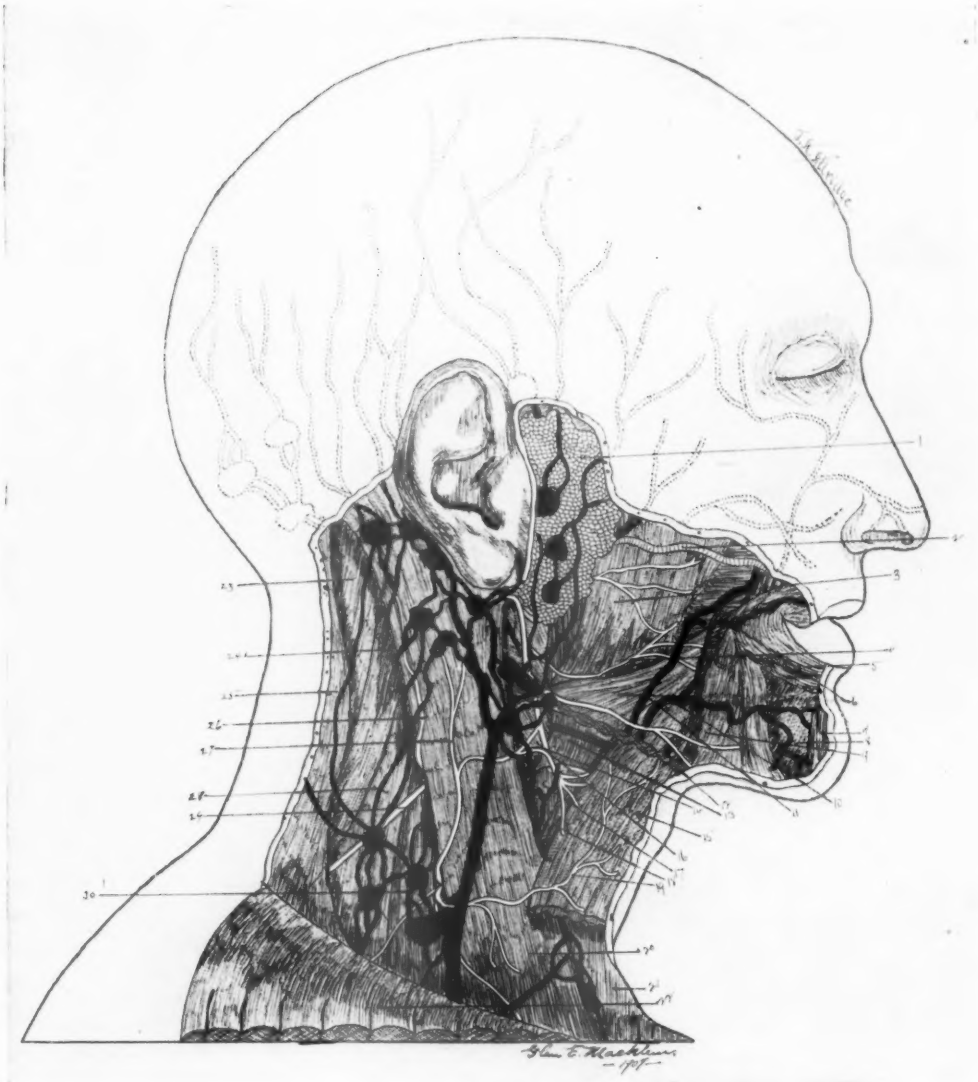

PLATE I

EXPLANATORY KEY

1. Parotid gland.
2. Stensons duct.
3. Masseter muscle.
4. Buccal nerve.
5. Orbicularis oris muscle.
6. Buccinator muscle.
7. Levator menti muscle.
8. Depressor menti muscle.
9. Depressor anguli oris muscle.
10. Facial artery.
11. Facial vein.
12. Upper cut edge of platysma.
13. Hyo-glossus muscle.
14. Infra-maxillary nerve.
15. Platsyma myoides muscle.
16. Digastric muscle.
17. Thyro-hyoid muscle.
18. Omo-hyoid muscle.
19. Superficial cervical nerve.
20. Sterno-cleido mastoid muscle.
21. Sterno-hyoid muscle.
22. Lower cut edge of platysma.
23. Splenius muscle.
24. Levator anguli scapulae muscle.
25. Trapezius muscle.
26. Occipitalis minor nerve.
27. Auricularis magnus nerve.
28. Scalenus posticus muscle.
29. Spinal accessory nerve.
30. Scalenus anticus muscle.

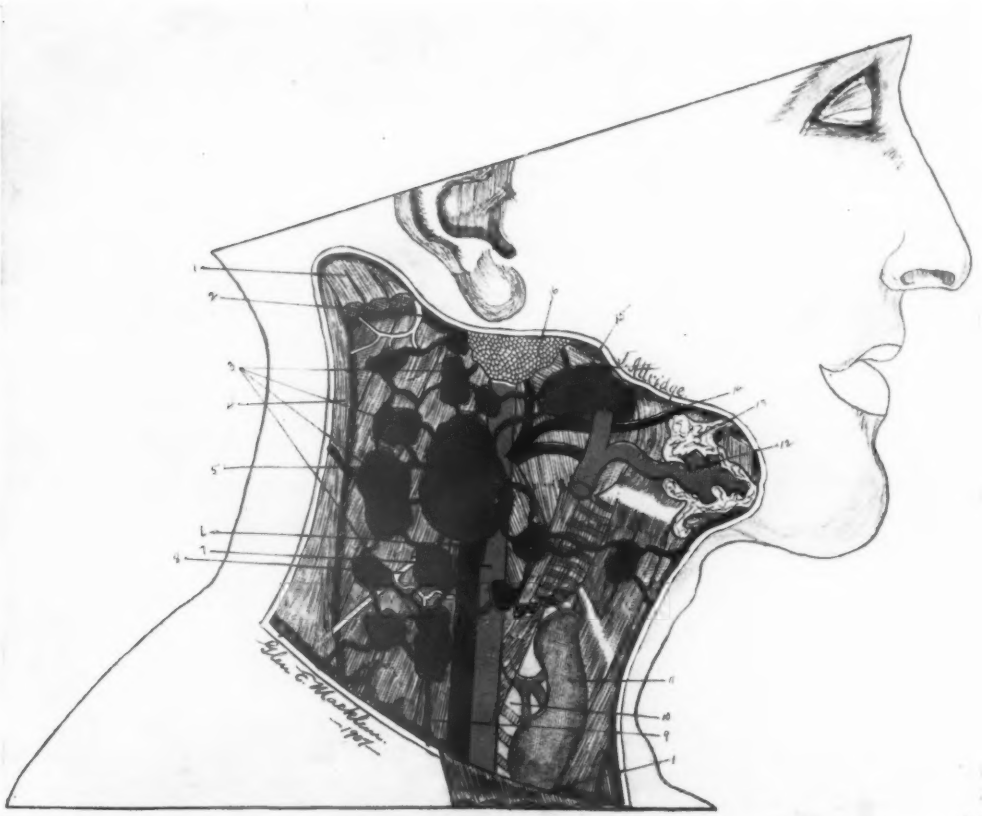

PLATE II

EXPLANATORY KEY

1. Sterno-cleido mastoid muscle.
2. First cervical nerve.
3. Internal substernomastoid glands.
4. Trapezius muscle.
5. Tonsillar lymph gland.
6. Internal jugular vein.
7. Common carotid artery.
8. Spinal accessory nerve.
9. Jugular lymph trunk.
10. Trachea.
11. Thyroid gland.
12. Facial artery.
13. Submaxillary salivary gland.
14. Facial vein.
15. Faucial tonsil.
16. Parotid gland.

On reaching the spinal accessory the glands should be passed underneath and the dissection carried up, removing all the glands in one mass, when possible. When all the glands cannot be reached from under the sterno-mastoid, this muscle should be divided in such a way as to save the nerve supply to it. The glands removed, the muscle should be sutured.

The submaxillary and submental glands are occasionally infected, when an additional incision, running across the hyoid bone in the skin creases to the middle line, will be necessary. In this location the inframaxillary division of the seventh (Plate 1), the hypoglossal (Plate 4), and the lingual artery and vein (Plate 4), should be searched for and avoided. Facial paralysis, of long standing, results from division of this nerve. Injury to the hypoglossal shows immediately in the tongue movements (Mayo). If this incision is made three-fourths of an inch below the angle of the jaw, it will miss the dangerous area. In the submaxillary triangle the facial artery and vein should be

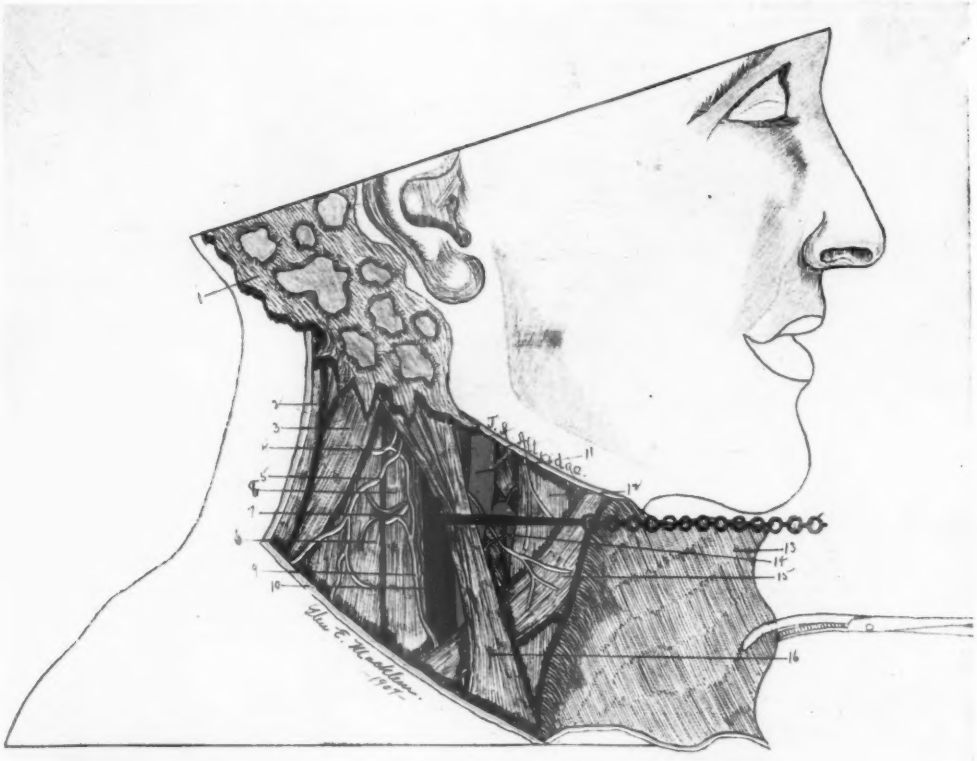

PLATE III

EXPLANATORY KEY

1. Excised lymphatic tissue.
2. Trapezius muscle.
3. Levator anguli scapulæ muscle.
4. Third cervical nerve.
5. Spinal accessory nerve.
6. Scalenus posticus muscle.
7. Fourth cervical nerve.
8. Scalenus anticus muscle.
9. Phrenic nerve.
10. Internal jugular vein.
11. External carotid artery.
12. Thyro-hyoid muscle.
13. Platysma and skin flap.
14. Superior thyroid artery.
15. Omo-hyoid muscle.
16. Sterno mastoid hooked back.

isolated. Where the glands are very large it is surprising how large the facial vein appears, probably due to pressure.

The phrenic and pneumogastric nerves may be injured in the middle of the incisions when the sterno-mastoid and deep jugular vein are retracted inward.

The lymph duct is occasionally injured, but usually heals in a few days.

When the parotid nodes, (Plate 1), are involved the infection may come from the middle ear, the face, or from the deep chain due to a damming back from the infected glands below. Here Stenson's duct and the facial nerve must be kept in mind.

The incision through the skin should run parallel with Stenson's duct (Plate1), and the glands be removed by blunt dissection.

On account of the extensive traumatism in a field so rich in absorbents, drainage is necessary. A small cigarette drain, suitably placed will be sufficient.

The wound is closed by suturing the platysma with running catgut and the skin by subcuticular suture. Suturing of the platysma prevents scar spreading.

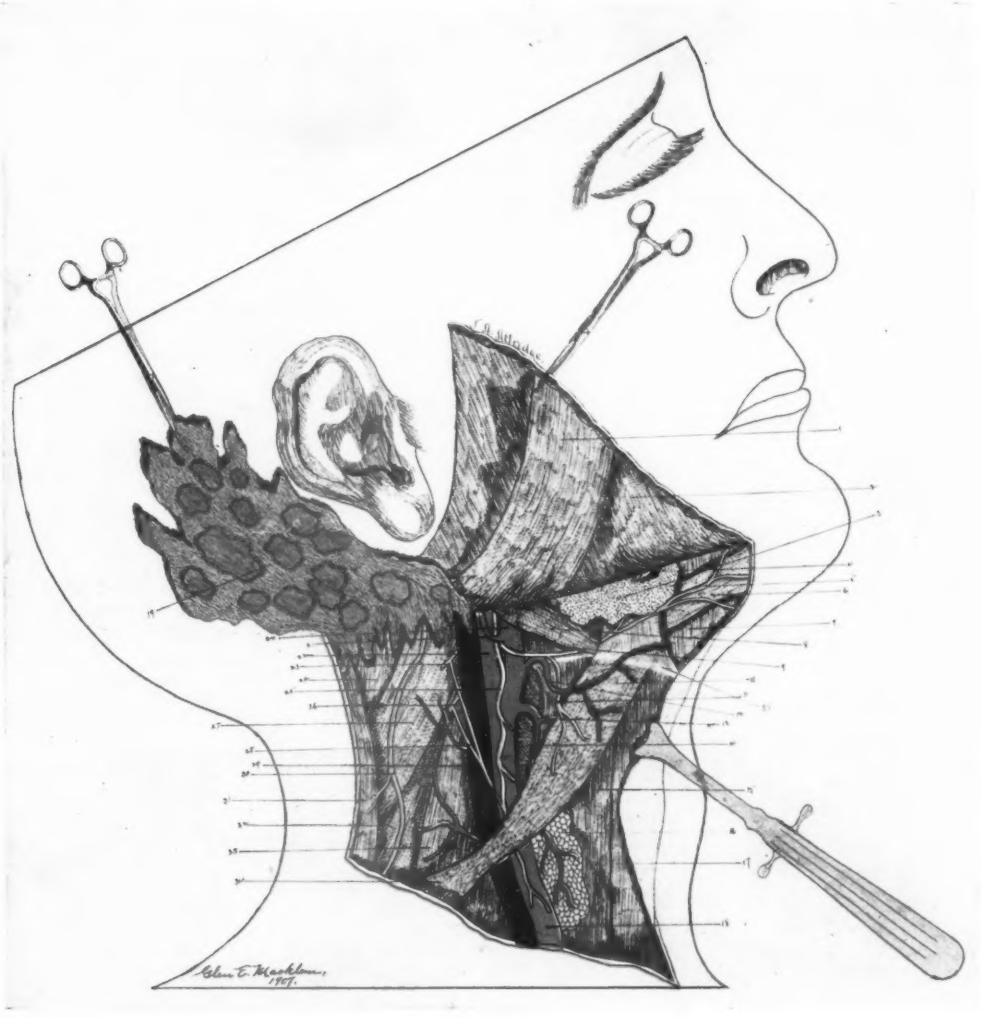

PLATE IV

EXPLANATORY KEY

1. Sterno-mastoid muscle.
2. Platysma myoides muscle.
3. Submaxillary gland.
4. Facial vein artery and nerve.
5. Glossal and mental nerves.
6. Digastric muscle.
7. Mylo-hyoid muscle.
8. Stylo-hyoid muscle.
9. Hyo-glossus muscle.
10. Anterior belly of omo-hyoid.
11. Thyro-hyoid nerve.
12. Lingual artery.
13. Superior thyroid artery.
14. Inferior constrictor muscle.
15. Thyro-hyoid muscle.
16. Thyroid gland.
17. Sterno-hyoid muscle.
18. Common carotid artery.
19. Excised mass of lymphatic tissue.
20. External carotid artery.
21. Occipital artery.
22. Internal carotid artery.
23. Second cervical nerve.
24. Complexus muscle.
25. Descendens hypoglossi nerve.
26. Third cervical nerve.
27. Splenius muscle.
28. Communicans hypoglossi nerve.
29. Internal jugular vein.
30. Spinal accessory nerve.
31. Trapezius muscle.
32. Levator anguli scapulæ muscle.
33. Scalenus anticus muscle.
34. Posterior belly of omo-hyoid.
35. Hyoid Bone.

Some of the cases operated upon will need a second operation, the number depending on the thoroughness of the first operation, and on the power of the anti-tuberculous bodies to cope with infection unavoidably missed. In a series of forty-eight cases treated in St. Mary's Hospital, N. Y., one patient was operated upon eight, and another ten times; some operations were for extirpation, some for incision.

The patient on leaving the hospital should be advised how to live. He should be asked to report for inspection in three months, and at longer intervals, as conditions indicate. He should be told that other glands may enlarge, and if so, should be removed, and finally that such glands are not recurrences, but glands that were not recognizable at the time of operation.

CONCLUSIONS

The points favoring early and thorough removal of tuberculous glands, in suitable cases, are:

1. The high percentage of recoveries, as compared with other lines of treatment.

2. The great tendency to metastasis according to statistics. One-quarter to one-half of the cases not so treated develop tuberculosis of other organs.

3. The persistence with which patients react to tuberculin test after prolonged out-door life, though otherwise apparently improved, showing. that the bacillus is still present and viable.

4. The comparative safety of the operation. Dowd, Jordan, Wohlgemuth, Blos, and Mayo report respectively: 134, 429, 167, 328, and 500 without mortality.

The points against operation are:

1. Deaths do occur: From prolonged operation, consequently, if both sides are involved, requiring extensive dissection, two operations should be done with an interval for recuperation between them. From hæmorrhage, which can usually be avoided by careful technique.

2. Extensive involvement of internal organs.

After a successful operation, patients should be put into the open air as soon as possible and taught how to live, not forgetting the use of reconstructive medical treatment and the necessity for careful medical supervision.

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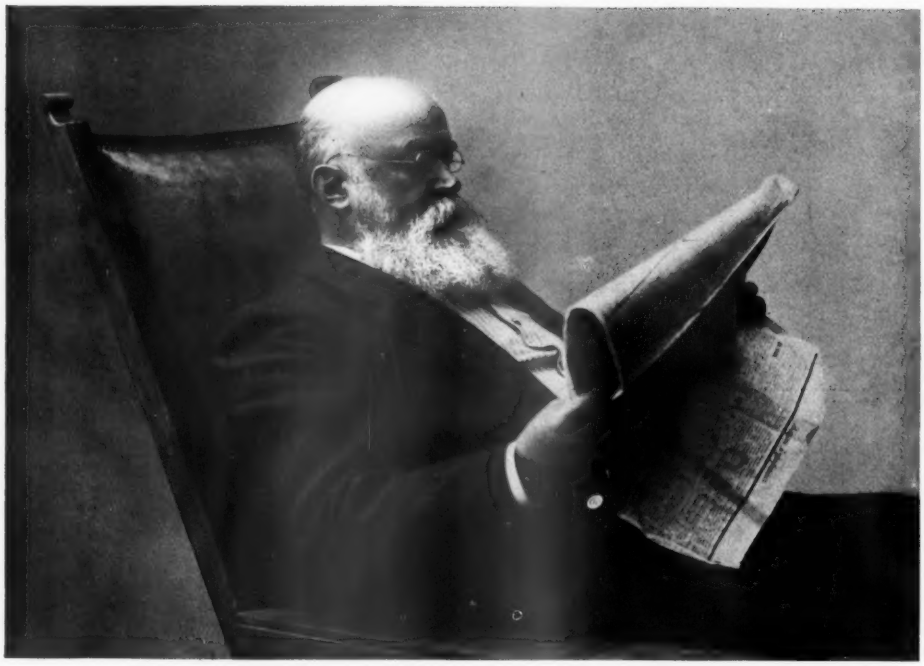

PROFESSOR ORDINARIUS P. J. DIAKONOW

DEPARTMENT OF
NICHOLAS SENN FESTSCHRIFT

A CONTRIBUTION TO THE QUESTION OF EXTROPHY OF THE URINARY BLADDER

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THE disease, or rather the condition which we designate as extrophy of the bladder seems to be in different ways still unclear. The solution of its etiology and pathogenesis is only hypothetical as these hypotheses present no sufficiently complete explanation of the conditions present.

The pathological anatomy is not yet sufficiently investigated as the number of autopsies upon patients suffering from this malady is very small; Professor Enderlen[1] in 1904 was able to collect only 16 cases.

The examination of a larger amount of pathological material would not completely familiarize us with the subject since the forelying changes in the different cases signalize such great diversity, in consequence of which it is difficult or well-nigh impossible to speak of a rational treatment of bladder extrophy.

On the other hand, the condition of such patients is so grave, the possibility of complications dangerous to life so great, the maintenance of the necessary cleanliness and proper nursing so difficult that the surgeon, in the face of these difficulties, can and dare not remain simply an observer. When the possibility of a rational therapy based upon etiology and pathology is not known we have a multiplicity of rules of procedure against dangerous and troublesome symptoms. It is in this direction that our efforts at present are directed, and those symptoms which seem most dangerous to the patient, and against which the many and generally employed rules of operation are directed, are in the first place, the unprotected and open position of the bladder mucous membrane with the openings of the ureters, and the incontinence of urine.

When we unite the foregoing conditions into one problem and add thereto the various phenomena occurring at the site of the operation, we must recognize, that in spite of these important restrictions, our means are not abundant.

The number of operations together with their various modifications suggested for extrophy of the bladder is so large, not perhaps because the treatment is so worked out in detail and the different operations corresponding to the different conditions found; on the contrary, the large number of proposed operations is, as in some other conditions, only an evidence of our helplessness and the failure to reach the goal set up by these various procedures.

Such conditions compel us always to seek new and more complete methods

[1] Prof. Enderlen — Ueber Blasenektopie, Wiesbaden, 1904, L. 60.

and prompt us to publish the practical application of the method which has been worked out and satisfactorily used by us. When we consider the formation of a new urinary receptacle which will retain the fluid and bring its evacuation under the control of the patient's will, we naturally turn first to the closely related intestine, and we find this to be the ground principle in many cases where an operation for the cure of extrophy of the bladder has been done, viz., the conduction of the urine into some portion of the intestine, even to the formation of an artificial bladder from a part of the gut.

The unfavorable conditions occurring in all these cases lie in the possibility of the passage from intestine to ureter and thence to the kidney, of the contents of the intestinal canal which none too seldom leads to a dangerous kidney affection. In order to avoid this condition Maydl proposed his well-known operation of transplanting a goodly portion of the bladder mucous membrane surrounding the ureteral openings into the rectum, hoping in this way to retain the normal mechanism of the parts and in a normal way to prohibit the return of the urine to the ureter and kidney. The procedure is undoubtedly rational and the Maydl operation has become widely known and quite generally practised.

By careful consideration one cannot overcome some doubts as to the truth of this hypothesis.

First: We do not know if the lower end of the ureter pursues the same course within the walls of the ectopic bladder as in a normal one.

Second: We know that the openings of the ureters in the normal bladder tend to close by pressure and expansion due to accumulation of fluid within the bladder.

The ectopic bladder on the other hand, is never distended, in consequence of which the transplanted portion of bladder surrounding the ureters will fail in the valve-like action, lacking as it will, when transplanted into the bowel, the pressure due to accumulation and globular expansion. All these considerations have been observed in patients who have undergone the Maydl operation. A certain percentage of these cases have sooner or later after the operation succumbed to kidney disease.

Professor M. S. Subbotin brought before the Russian Surgical Congress at Moscow, in 1900, a new advancement in the treatment of extrophy of the bladder which is reported in the Zentralblatt für Chirurgie No. 51, s. 1257, therefore I shall not go into detail; suffice it to say in general that a communication between the ectopic bladder and the rectum is made, and from the mucous membrane of the rectum a canal is formed which serves as a reservoir for the urine. Increase of pressure serves to expel the urine, while contraction of the sphincter serves to close the canal (see A, Fig. 1). The last act of the operation is the closure of the opening above the pubes through which the bladder mucous membrane protrudes, by means of one of the many plastic procedures.

The urine is, after the Subbotin method, as after the Maydl operation, delivered into the large intestine but into another part of it, viz., the rectum, which in every respect seems suitable.

By this operation also a mechanism is constructed which does away with the possibility of infection of the kidney by intestinal contents. This mechanism is not so problematical as in the operation devised by Maydl; in fact, by a properly conducted operation as done by Subbotin a kidney infection by intestinal contents is impossible.

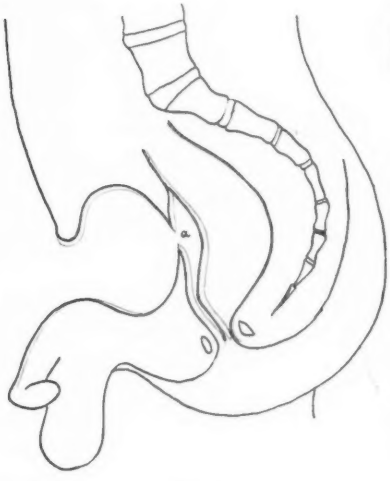

Fig. 1.

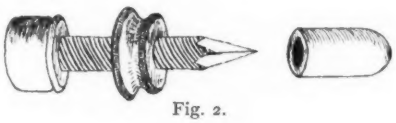

Fig. 2.

I have done the Subbotin operation several times and am convinced of its leading advantages, but I must say that it must be classed as one of the most difficult. Usually, it is not possible to arrive at the end result by means of two operations, as in the after-treatment different corrective and supplementary operations are necessary according to the different conditions which may exist in the different cases; the end result is reached only after a lapse of several months and in all cases social and family conditions will not permit of such long treatment.

In order to hasten and simplify the first part of his operation, Prof. Subbotin recommended an instrument (see Fig. 2), a heavy, nail-like, threaded metal bolt with a nut and cap covering its sharp point. The instrument is allowed to remain *in situ* until the crushed tissue becomes necrotic and adhesions have formed between the apposed tissues. The instrument then comes away leaving a communication between the bladder and rectum. Professor Subbotin, however, was not satisfied with this simplification, and further proposed a new operation which he in a letter to me described as follows:

1. Transverse incision, anterior to the anus extending down to the external sphincter.

2. Blunt dissection of the anal wall from the external sphincter.

3. Passage of a curved trocar from the wound through the soft parts, the point of emergence being the trigonum below the ureteral openings.

4. Withdrawal of a No. 26 rubber catheter by means of the trocar through the canal made by the passage of the trocar. This drain is left in place until the canal is marked by cicatricial tissue and represents the newly made urethra.

5. Formation of an anterior bladder wall and closure of the abdominal wound. In my last two cases I utilized for that purpose the rudimentary penis as it seems with good results.

6. In girls the uterus must first have been removed.

This modification of Subbotin's original operation simplifies greatly the treatment of extrophy of the bladder. In my judgment, the weak point in the operation lies in the fact that the newly formed urethra is not lined with epithelium, in consequence of which the walls will soon unite, the canal then ceasing to be patulous.

It appears to me to be more important to begin the operation with a dissection of a quadrangular flap of mucous membrane whose base lies immediately at the ureteral openings. The trocar is then passed through from below emerging not below but rather a little above the ureteral openings. The flap of mucous membrane is now ligated to the trocar and drawn down into

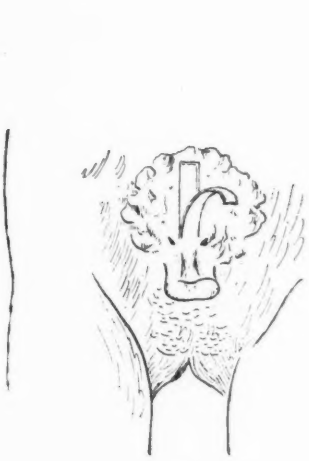

Fig. 3.

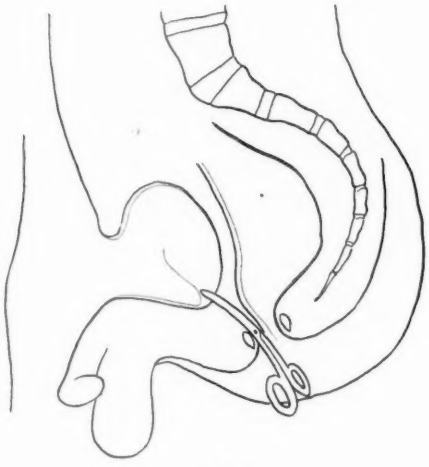

Fig. 4.

the canal made by the passage of the trocar. By this means we have urethra lined with bladder epithelium. It was by this modification of Subbotin's operation that I treated the following case.

The child, a five months' old boy with extrophy of the bladder, aside from non-union of the symphisis pubis, and a double inguinal hernia, showed no signs of faulty development. The nutrition was insufficient. The operation, which was made on June 28th, 1907, was easily and quickly done, and I, as well as the visiting physicians, was impressed with the favorable outlook of the case. After the withdrawal and retention of the slip of mucous membrane to the skin of the anus, there presented at the base of the bladder a funnel-shaped depression into which the ureters opened, and in consequence of which emptied the urine into it in spite of the fact that the bladder in front of it remained open.

During the entire after-treatment the greater part of the urine escaped through this newly built urethra in which a rubber drain was allowed to remain.

Unfavorable conditions developed the evening of the day of operation. The temperature rose to 39.4° C. and remained high throughout. The first day after the operation the patient relished his food but began to suffer from diarrhœa which was followed by obstipation with tympanites. On the fourth day, dullness of intellect developed and the patient died on the fifth day of July, eight days after the operation. Since no autopsy was held, I would rather refrain from expressing an opinion as to the operation's being wholly or in part concerned in the direct cause of death.

We may have had to deal with an incidental complication, viz., acute gastroenteritis such as often occurs in such young children at this season of the year. On the other hand, it may have been an acute peritonitis caused by the trocar which was passed "in the dark" so to speak. We do not know for a certainty how deeply, in cases of bladder extrophy, the fold of peritoneum extends between the bladder and rectum. Such an injury to the peritoneum is certainly not conjectured, but such might have been the case. It is desirable to rule out such possibilities when one must operate in the dark, which procedure from the standpoint of modern surgery is not to be recommended.

This experience leads me to recommend a modification of the operation, viz.:

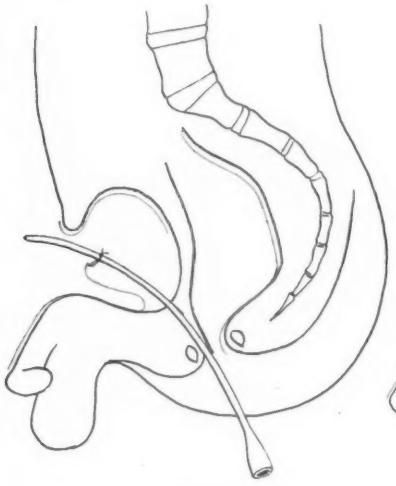

Fig. 5.

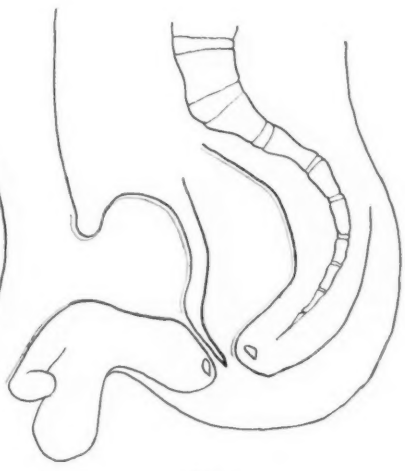

Fig. 6.

1. Dissection of the longest possible quadrangular flap of bladder mucous membrane whose base is directly at the openings of the ureters (see Fig. 3). The flap may be in the middle line as in the figure or it may be oblique in order to gain length, or if necessary it may be extended over the border of the mucous membrane into the skin.

2. Through a 2 cm. long, transverse incision in front of the anus, the mucous membrane of the rectum together with the submucosa is separated from the external sphincter, the dissection being made with a scalpel to a depth of about 1.5 cm. and continued with closed blunt shears through the tissues to the bladder, the shears being controlled by a finger in the bladder (Fig. 4).

3. Upon puncture of the bladder by the shears a rubber catheter is passed from below and upon the end of the catheter the end of the flap of mucous membrane is made fast by means of a silk ligature (Fig. 5). By withdrawal of the catheter the flap of mucous membrane is drawn down through and sutured to the skin of the anal border. The result is a newly built urethra having epithelial walls which at the will of the patient can be opened or closed by the action of the external sphincter (Fig. 6).

4. As a second act of the operation follows the closure of the anterior abdominal cleft either by direct suture of the edges or by utilization of one or another plastic operation according to the requirements of the case in hand.

The following operation was done in the manner described.

On September 17, 1907 a twenty-months-old boy was brought into my clinic. The family history reveals no trace of disturbed development. The child was well nourished, weighed 29½ pounds, length of body 81 cm. The internal organs were normal. The mucous membrane of the bladder presented a rounded tumor elevated about 14 cm. above the surrounding surface and about 5 cm. broad. In the center of the tumor two papillomatous areas extending about 1 cm. above the surface presented, in the centers of which the ureters opened and from which by increased intraabdominal pressure, urine was expressed. The entire surface of mucous membrane was covered with excrescences giving it a cauliflower-like appearance. In the upper part of the bladder was a small scar-like area which corresponded to the umbilicus.

The lower part of the bladder mucous membrane was continuous with the mucous membrane of the urethra, constituting epispadias. The scrotum contained the undeveloped testicles, which at times slipped upward, and occupied the inguinal canals. There existed a left inguinal hernia, also a separation of the symphysis pubis.

On September 21, 1907, the operation was made in the manner already described. On account of the small area of bladder mucosa the flap was increased in length by extension of the incisions into the skin. The cicatricial tissue at the border of the bladder was so thin that the peritoneal cavity was opened and a loop of intestine was extruded. This was immediately replaced and the peritoneum closed by a purse-string suture.

The defect in the bladder mucosa due to the dissection of the flap was closed by sutures and a catheter was then placed in the newly made urethra. The condition after the operation was favorable, the transplanted flap remained viable in its new position, and after one month had elapsed the second act of the operation, viz., the closure of the anterior bladder wall was undertaken. On October 21, 1907, the edges of the bladder wall were freshened and sutured together, and although no difficulties were encountered, the sutures, even on the first day after operation began to cut out and soon the whole mucous membrane was exposed.

A second attempt at closure was undertaken on November 1, 1907. Beneath the penis two slightly curved incisions were made leaving a skin space 4 cm. broad between them. The skin was then dissected up and the result was a bridge-like flap which with the loose tissue beneath it extended toward either side. The skin on the under surface of the penis was divided in the median line and dissected up on either side. This made two skin flaps between which was the denuded posterior surface of the penis. The flaps were reflected over the mucous membrane of the bladder and after freshening the edges of the bladder were sutured into place. The bridge flap was then sutured in place over the denuded penis.

Four days after the operation all went well and it seemed as if a primary union would result; the entire amount of urine passed through the new urethra. On the fifth day a small amount of urine came through at the site of the sutures; after a week the sutures were removed and it was found that in the upper part of the wound, union had taken place very sparingly; the lower part of the wound however, was completely healed. Another operation will be necessary to complete the case.

As the mother had remained in the clinic the entire time and could stay no longer, she took the child away with a promise to return in a short time and have the operation completed. A hindrance to healing was found during the second operation, when, by freshening the edges of the skin already eczematous and wet with urine, it was seen that the cicatricial tissue would be slow to heal; however, this was to be expected and nothing irreparable was to be seen.

Plastic operations upon these parts in such cases always demand secondary interference and I do not doubt that in this patient I can attain a very favorabe result.

The principal part of the first operation as above described and which was easily accomplished was the formation, at one operation, of a useful urethra which is under control of a sphincter. This was also attained in the first case described and in spite of the open bladder most of the urine was carried away by the newly formed urethra. In the second case the entire amount of urine was passed through the urethra so long as the closure of the anterior bladder wall remained complete. My experience with these cases warrants me to recommend this operation as technically easier and more certain of favorable results than the other, both of them having the same result in view. It will be seen that this operation is only applicable to extrophy of the male bladder as it is evident that the operation upon the female would be much more difficult.

Professor Subbotin recommends the removal of the uterus. At present, I prefer not to decide just how much advantage the application of these principles, as done in the male operation, after the removal of the uterus would outweigh the disadvantage of such stringent measures as the removal of the uterus.

TRANSACTIONS OF SOCIETIES

MISSISSIPPI VALLEY MEDICAL ASSOCIATION, OCTOBER 14, 1908

Discussion of the Symposium on Pancreatitis

Dr. G. W. McCaskey, Fort Wayne, Indiana: Until very recent times the diagnosis of pancreatic disease presented practically insurmountable difficulties, and I cannot refrain from saying that to American medicine belongs the credit for the most important fundamental advances that have been made in this subject. For these facts we are indebted to two men, and without the work of those two men a chapter on the pathology of the pancreas could not be written. I refer to the work of Fitz and Opie.

In going over the subject of pancreatic disease generally, it seems to me to be useful to us as clinicians to take a sort of bird's-eye view of the subject and divide all pancreatic diseases into three groups. First, the fulminant type, the hæmorrhagic, the acute suppurative conditions to which reference has been made by the surgeons particularly, and which are especially of a surgical character so far as treatment is concerned. Second, organic cases of the subacute or chronic type. Third, a group of cases which have received, I think, altogether too little attention, those of a purely functional character, with possibly organic bases of some sort, perhaps unrecognized transient pancreatitis.

One of the most important results in the whole study of the relationship between gall-stone disease and pancreatitis, it seems to me, is the much greater importance which has been given to gall-stone disease. Its relationship to pancreatitis has been clearly demonstrated by the statements made here to-day, and we should remember that it is necessary for a gall-stone to reach the diverticulum of Vater and block up its duodenal orifice in order to produce pancreatitis. Anatomical studies have shown that in sixty-two per cent of all cases the common duct was more or less firmly embraced by the pancreas. It is perfectly obvious that a calculus in passing through the intrapancreatic portion of the common duct may press upon the pancreatic duct itself, thereby producing stasis of pancreatic secretion and leading secondarily to chronic pancreatitis. It is only necessary for this pancreatitis which is at first interstitial in type, to proceed to involve the islands of Langerhans, with the production of diabetes or glycosuria, and perhaps finally leading to the complete syndrome of diabetes.

Dr. Croftan seems to cast some doubt upon the reliability of glycosuria as an indication of pancreatic disease. Certainly we can have, as he says, extensive disease of the pancreas without glycosuria. If we have glycosuria we can suspect that it is due, in many cases at least, to interference with the pancreatic function.

The close relationship between hepatic and pancreatic disease leads, as has been pointed out to-day, to difficulties in differentiating the two diseases. I will call attention to one point, namely, I do not think we can tell when we get a glycosuria whether it is due to hepatic insufficiency, to failure of the glycogenic function of the liver, or to the glycolytic function of the pancreas. If, instead of getting glucose, we get levulose, and the latter passes through the liver unchanged and appears in the urine, it is due to or is of hepatic origin. If the reverse is the case, it is probably pancreatic in its character, and the liver is entirely competent.

I wish to call attention to a third group of cases, the cases of pancreatic insufficiency without the severer well-recognized facts of pancreatic disease. Smith has recently published a series of cases, very chronic in character, in which he has assumed a condition of achylia pancreatica, either partial or complete, as the result of disease which was entirely remediable in character. If that is true the diagnosis of pancreatic insufficiency becomes of the first importance, because these cases were amenable to treatment, and several of his cases, which lasted over a long period of time, accompanied with diarrhea, emaciation, and the like, had entirely yielded to treatment. For us internists particularly, the problem of pancreatic insufficiency is the one great problem. I wish to say right here, however, I do not believe that any test of pancreatic insufficiency will enable us to diagnose with a strong degree of probability the existence of localized organic disease, such as carcinoma, in the early stages, because nature

has been prodigal of function, and we have much more pancreas than is needed, and a considerable portion, perhaps one-quarter or one-third of the pancreas, can be destroyed without interference with its function; the hemolytic and proteolytic functions may go on perfectly with one-quarter or one-third of the pancreas lost.

What are the data available for the recognition of pancreatic insufficiency, or what has been termed achylia pancreatica? Lepine, in a valuable contribution, to which Dr. Ochsner has referred, says after an extensive study of the literature, including his own personal experience, that he lays stress upon three facts as being the most reliable signs of pancreatic insufficiency: First, the accumulation of fat in the stools — steatorrhea; second, interference with nuclear digestion; third, a reduction in the quantity of ethereal sulphates in the urine. I agree with Dr. Croftan that the presence of steatorrhea is an unreliable sign of pancreatic disease. We must not forget our physiology, which has been referred to. We must remember that the bile has almost as much to do with the assimilation of fat as the pancreas. If pancreatic secretion is eliminated from the intestinal tract, the bile is still capable of affecting a considerable proportion of fluid, say fifty per cent of increased assimilation under these conditions. If the bile is eliminated and the pancreatic secretion remains, when we find steatorrhea we may say that we have a choice between the existence of pancreatic disease or hepatic disease, or both, and it becomes a question of weighing all the evidence and considering all data in order to determine, which or both.

In regard to some of the other tests, I will not take time to refer to Cammidge's further than to corroborate what the gentleman from Cincinnati (Dr. Schroeder) has said, and also what Drs. Mayo and Ochsner said regarding this complicated and tedious test. I have been using this test in my laboratory for two or three years, and while it is of considerable value, it is too cumbersome and complicated for routine use. I failed to recognize one case of pancreatic disease because I did not use it in every case of suspected abdominal disease. The patient was a woman, fifty years of age, whom I saw in consultation in an adjoining town, with a tumor on the left side, severe abdominal pain, which developed gradually until there were nodular growths in other parts of the abdomen. I did not suspect nor did I diagnose pancreatic disease. The patient was brought to Fort Wayne, placed in a hospital, and operated on by my surgical colleague, Dr. Porter. At the operation we found extensive fat necrosis and a fairly large tumor in the right abdominal area, due to vast accumulations of fat necrosis in the perirenal fat. That was Dr. Porter's opinion at the time of the operation. Had the Cammidge test not been so complicated, and taken so much time, and had it been used as a routine procedure, probably it would have given us an idea of the nature of the case prior to the operation.

The nuclear test, I believe, is of great value, provided it gives us a positive result, and if we follow Mueller's method, of giving a meal, and then a calomel purge in a couple of hours, when the pancreatic function is active, I think we will get a more reliable result. The absence of trypsin in the feces under ordinary conditions does not have great value unless we have the conditions under control, but if we can establish the fact that there is failure on the part of the intestinal tract to carry on nuclear digestion, it is strong evidence of pancreatic disease.

So far as the general subject of pancreatic disease is concerned, particularly with reference to the diagnosis, whileI agree with Dr. Croftan, I do not feel as pessimistic as he does. However, the diagnosis presents difficulties of a grave character. Taking the pathological foundation established by Fitz, Opie, and others, and the invaluable aid we have received from surgeons who have given us the value of their results and experience, and supplementing this by the laboratory methods to which reference has been made, I believe we can in a large proportion of cases make a very probable diagnosis of pancreatic disease, and I would make a special plea for the recognition of transient conditions of pancreatic insufficiency, which I believe exist, just as we have cases of gastric insufficiency existing without serious gastric lesions of any sort whatever. In fact, I would plead for thinking of pancreatic disease momentarily in the treatment of gastric disease. We know that we have cases of weak digestion, of low acidity, where there is very little hydrochloric acid present in the gastric juice, and the digestion is weak, is easily upset, and have we not similar cases in connection with the pancreatic function?

I feel inclined to agree with Dr. Ochsner in regard to the point that attacks of indigestion occur in chronic cases of pancreatic disease, and are the result of gall-stone colic. If they are attacks of indigestion — and I believe that is the term the Doctor used — I am inclined to think they are due to transient conditions of pancreatic insufficiency.

I wish to express my thanks to the gentlemen who have presented papers in this admirable symposium on this practical and important subject.

DR. WILLIAM H. WATHEN, Louisville: While we have been told much, we will naturally come to the conclusion that we have at this time only opened the door that leads us into labyrinth that is unknown and must be explored. We certainly do not understand the physiology oi the pancreas when it comes to the formation of the enzymes that aid in intestinal digestion, and the enzymes that aid in metabolism. Other enzymes may be discovered, and we may now believe that we have discovered facts in many particulars that are not true. Original research has done much to aid us in this work. We have learned, however, one thing, that while we cannot positively diagnosticate pancreatitis, we can diagnosticate a condition in the right upper quadrant of the abdominal cavity that indicates an operation, and by an exploration we will find a pathological condition that will need surgical attention. Let that be what it may, the patient is benefited.

It is a question whether pancreatitis is always the result of infection ascending from the duodenum, or through the blood, or whether it comes down secondarily from the gall-bladder; but the treatment is practically the same, and that is drainage. The best method of drainage in the chronic forms is through the biliary ducts, either by cholecystostomy, which will cure many of these cases, whether there be gall-stones or not, and I have had a number of cases recently with no gall-stones which were cured by drainage, or by other methods. But the ideal treatment, as presented by Dr. Mayo, would be, where complications do not contraindicate it, a cholecystenterostomy, preferably with the duodenum, next with the jejunum, as high up as is consistent with good surgery, and finally, unfortunately, it has been made with the colon, where we lose all the digestive properties of these secretions. I should advocate in all these cases operative procedures, drainage by cholecystostomy, or drainage by cholecystenterostomy into the duodenum or by the jejunum.

DR. FENTON B. TURCK, Chicago: I would like to add my little mite to the beautiful work that has been brought forward by Drs. Ochsner and Mayo, and the remarks that were made by Dr. McCaskey, by citing an interesting case which I recently had, and some investigations that were made in connection with sprue. These were two cases from China in which we made investigations of the pancreatic functions, and found that many English observers regarded sprue, or this form of tropical dysentery found in China, as due to pancreatic disease. We found by investigation that the phenylhydrazin test was positive; there was fat in the stools, with all evidences of disturbance of the pancreas, even of painful points. We made a careful investigation of all the facts we could obtain in relation to the symptomatology of pancreatic disease. We found, to make a long story short, that the infection in these cases was neither due to the anaërobia of Welch nor the bacillus enteritidis of Klein. It was between the two. We could not grow these bacilli by the ordinary method, and therefore we adopted the following method: We injected, according to the method of Welch, feces into the jugular vein of a rabbit, killed the rabbit, incubated it for six hours, and we found when incubated for twelve hours destruction was so great and putrefaction so intense that we could not utilize the material. With six hours' incubation, we took a small part of the liver of the rabbit, rubbed it up in emulsing, and injected it into the intestines of a pig, having anaërobic conditions for the micro-organism, and it developed rapidly. We made a vaccine from this. Dr. Hektoen was called in consultation, so that we could feel warranted in using this for a vaccine. We found that if we heated the vaccine at 60° C., it destroyed its usefulness. We heated it at 60° C., and injected it into the patient. The number of stools per day varied from fourteen to sixteen. Remember, this patient had the disease two and a half years. One of the patients weighed sixty-one pounds, and the other sixty-three. After the third injection of the vaccine the stools were reduced from twelve to fourteen down to three or four a day. After the eighth injection the patient was rather constipated than otherwise, and we found that the pancreatic function inside of two weeks was restored. Now, the patient weighs 120 pounds, and the other one, 130 pounds. We have restoration of the function of the pancreas, together with the disappearance of the intestinal disturbance, and with it the anaërobic infection has also disappeared.

We are now carrying on further researches along this line. I present a report of these two cases as an illustration of the relationship between infections that may take place and such toxemias as affect the functions of the pancreas, with the restoration of such function through the elimination of the original infection.

DR. GEORGE DOCK, New Orleans: I do not like to break in upon the very illuminating impression that has been made this morning by all the speakers. As a general thing, I agree with all that has been said on both sides, and if I add to the symptom-complex that we have to consider in connection with pancreatic disease, I would not think of doing so to the exclusion of prompt treatment in other ways, but there is one symptom to which attention has not been called this morning.

It was pointed out some years ago, and has been noticed by a good many observers since then, that certain cases of acute inflammation of the adrenals produced the same symptoms as acute pancreatitis, or a great many other cases of acute irritation of the peritoneum, so-called peritonism symptoms.

Lavenson has reported an interesting case in which a patient had chronic pancreatitis and acute hæmorrhagic degeneration of the adrenals. In mentioning this it may be supposed to add to the difficulty of the diagnosis, but I would hasten to add my opinion in all such cases with acute symptoms, that laboratory methods of investigation are out of place. The surgeon settles the point there. I would suggest that in all cases of this kind a more careful investigation of the adrenals be made than perhaps has hitherto been the case, and I would enlarge this to the autopsy experience. I think in all such cases the adrenals ought to be more carefully examined than has been the habit of pathological anatomists before this.

There is a clinical symptom in adrenal disease which should be looked for more carefully, and if I understand Lavenson's article correctly, he has not put the matter as clearly as he might have done. Four years ago Sergent described a symptom of adrenal disease which he called a white line. The white line he perceived is not unlike that which we get in vasomotor spasm in spreading the finger over the skin. From experience in two cases of Addison's disease this interesting symptom, associated with a white line, can be looked for in many cases where it has not been done heretofore. The white line, as seen in these cases, is not the narrow white line we get in many cases by spreading with a sharp point along the skin, but looks more like the line we may make by spreading powder with broad surface over the skin and it acts on slight stimulation of the skin. In one of my patients, who died two months afterwards with Addison's disease, when one lifted the bedclothes, the sheet or nightgown, all the folds of the clothing had left that white line, making the skin look as though it had been strewn evenly with a white substance wherever the folds had touched. On stroking gently with the finger or with a smooth object, a line about a centimeter and a half formed, which remained for some time. In many cases this is a symptom of Addison's disease of some diagnostic importance. It is not supposed that it will be equally strong in all cases, but I mention it now because I think it should be more carefully looked for by those who examine patients either *in vivo* or at autopsy, or what may be most convenient for them.

DR. DANIEL N. EISENDRATH, Chicago: There is one point mentioned by Dr. Croftan and by Dr. Mayo which we cannot emphasize too strongly, and one which I believe we should take into consideration in the future, both the internist and surgeon, in the diagnosis and surgical treatment of disease of the pancreas, and that is the frequency with which we have primary pancreatitis, that is, primary so far as we know. Of course, these cases are secondary to infection from the intestines, but primary, so far as any gall-stones are concerned, in the pancreas. We have been accustomed in the past to think almost all cases of persistent and intense jaundice were due either to malignant disease at the head of the pancreas or to stones in the common duct, or to some similar condition obstructing the common duct, as tumors, pressure, and the like.

We have come to recognize that there is a so-called class of primary icterus which is due to inflammation of the pancreas resulting in such a proliferation of connective tissue around the orifice or close to the orifice of the common duct into the duodenum that there is persistent jaundice, and this jaundice will not be relieved by any efforts that are directed towards cholecystostomy, but these are the cases in which cholecystenterostomy or cholecystjejunostomy is the only operation which will give them relief, provided it is possible.

I saw an instructive case of this kind during the past few months and operated on it. The patient was a woman, who had been intensely ictaric for one year, with typical recurrent attacks. I laparotomized her. I found the gall-bladder, which was very much distended, contained two small stones. The next thing that attracted my attention in searching, as we usually do for stones in the common duct or disease at the head of the pancreas, for further causes of the icterus, and examining the common duct down to the ampulla of Vater and finding no gall-stones, was the pancreas itself showed intense chronic inflammation; it felt almost like a carcinoma. This was an example of a class of cases to which, I think, sufficient attention has not been directed in the past.

DR. NATHAN ROSEWATER, Cleveland, Ohio: I certainly can add nothing of interest or of great value to what has been already said, but to be brief, I wish to call your attention to one point brought out by Dr. Ochsner, namely, we have to eliminate gastroduodenal conditions from the pancreatic. Now, in the paper I presented yesterday, I made an effort to show how that can be done, and that is, by rest-cure of the stomach for one or two weeks in these chronic cases only. Where there is often great distress, by feeding through the rectum we avoid causing any func-

tional disturbance or interference on the part of the gastroduodenal tract, so in that manner I have in the cases I have seen, not a large number, been able to eliminate the gastroduodenal from the extragastroduodenal cases, and in that manner tried to distinguish between the two classes of cases.

DR. J. H. CARSTENS, Detroit, Michigan: It seems to me there are one or two lessons to be learned from these papers. Let us take the paper on the etiology of pancreatitis, and it shows that nearly all the cases start from gall-stones or are complicated by gall-stones. Although the infection may take place independently, it shows that gall-stones are generally the complicating factor in the cause of the trouble. If that is the case, it teaches us one great lesson, namely, that gall-stones are not such a simple, common, everyday thing as a great many physicians have heretofore thought. We have been told that the woods, so to speak, are full of gall-stones. In Germany they made post-mortem examinations, and demonstrated that gall-stones were present in at least twenty-five or thirty per cent of the cases. I have made post-mortem examinations, and for the last two years have examined every single case of laparotomy for gall-stones, and out of five hundred cases, I have only found two in which gall-stones were present. That shows to me that gall-stones are not so common as we have been told. It shows, furthermore, that when a person has gall-stones it is a serious affair, and it may be due to an infection or to pancreatitis. All cases having gall-stones should be carefully watched, and promptly operated upon. That is the point I want to make, and it is a good lesson for the general practitioner.

Take the other paper by Dr. Ochsner, and it teaches us a lesson, namely, that the laboratory is very essential in assisting us to make a diagnosis. Frequently I hear the laboratory pooh-poohed, but I must insist that physicians should be more careful and be taught how to make a diagnosis without the use of the laboratory. The laboratory, however, is certainly an aid to diagnosis in many instances. We need the clinical findings with the laboratory in order to make a correct diagnosis. Both of them should go hand in hand.

Patients with gall-stones should be operated on as soon after the diagnosis is made as possible. I had a woman come to me last week with gall-stones. She had had symptoms of gall-stone disease for six years, but all at once she began to lose flesh; she got weaker and weaker; she lost forty pounds inside of six weeks. Her doctor told her that she had a serious condition and must be operated on. She came to me and I found the pancreas very greatly swollen. Here was a case where a woman was said to have had gall-stones for six years without symptoms, and all at once some disturbance occurred, the pancreas began to be involved, she lost flesh rapidly, and on account of the good sense of the family physician, who understood the nature of these cases, she was operated on in time. I cannot emphasize too much the great importance of operating on these cases promptly, and if we do not know the exact nature of the trouble, we should open them up and find out what is the trouble.

DR. HAGGARD (closing the discussion on his part): Very little difference of opinion has been expressed in reference to the etiology, consequently I shall have only a word or two to say. We must not lose sight of the fact that, no matter what the etiological factors may be, there must be some injury to the architecture of the pancreas to cause the escape of the ferment that actually is necessary and is operative in the production of the fat necrosis which is the essential pathological change. No matter what it may be, whether gall-stones, the retrojection of bile, injury, or what not, there must be interference with the gland as well as the parenchyma itself to let out this ferment to activate the pancreatic juice in its destructive process. In cases of pancreatic diabetes, we must not lose sight of the fact which is well known that it is only the interacinous forms that really give rise to the glycosuria, and the practical lesson is this, that inasmuch as the interlobular forms precede the interacinous forms, there is ample time then, after the condition is recognized, before the supervention of glycosuria and diabetes, to remove the offending cause, we will say, gall-stones or infection, then we will escape the supervention of the interacinous form with the diabetes. Although Robson has cured some of these incipient cases of diabetes by operation and drainage, unfortunately the late cases have gone on, and while they have been temporarily relieved, they have died like all other cases of diabetes.

DR. SCHROEDER (closing the discussion on his part): I have very little to add to the paper I have read. However, I want to say one thing, namely, I have made particular reference to the Cammidge test, but I do not wish to say that this test will enable one to demonstrate positively whether or not he has disease of the pancreas to deal with, as probably no laboratory test can establish such a fact.

As to the importance of the diagnosis of pancreatitis, it is well and good to say you have a sur-

gical condition in the upper part of the abdomen, and therefore, you go ahead and operate. We have heard from Dr. Mayo that all the cases of gall-stone operations that do not do well are the ones that are complicated by pancreatic disease, and therefore, any method, laboratory or otherwise, that gives us any information on this subject, should have its value.

As to the treatment of the condition, the treatment is very largely that for the removal of gall-stones or inflammation of the gall-bladder, and there are, as a rule, three things that deserve attention. One is the obstruction, the other is the mechanical result of the inflammation, and then the disinfection of the gall-bladder. It seems to me the same things hold good in pancreatic inflammation.

No one has made reference to the method of treatment suggested by some other men. At the Johns Hopkins they have suggested that if urotropin is given in sufficiently large doses, it is excreted through the bile duct as well as through the ducts of the pancreas. I have used urotropin in large doses, that is, one dram given four times a day, giving it for a week at a time, and then stopping it for a time, because these patients complain of irritable bladder if the treatment is continuous, and I cannot say what the results will be in those cases at this time, but I hoped that some one else might make reference to this method of treatment.

DR. CROFTAN (closing the discussion on his part): I want to defend myself against the accusation of my good friend Dr. McCaskey, of being a diagnostic pessimist, for that is the worst thing I have ever been called. If the internist is to be, first, a therapeutic nihilist, and then a diagnostic pessimist, there is very little left for him to be.

I want to make this point, and it has been brought out by other speakers, that pancreatitis is so dangerous a disease that I would for once in my life, if I had a bad stomach ache and other dangerous symptoms, allow some one to make a laparotomy on suspicion for general results rather than wait for the working out of these finer laboratory methods. As I have said, if I had a bad stomach ache, I would go to Rochester, Minnesota, to Nashville, or Chicago, and not to Fort Wayne, and have some eminent surgeon make a hole in my abdomen in order to find out what was the matter.

DR. MAYO (closing the discussion): I always like to see Dr. Carstens with a brick in each hand because he hits the mark and it is a good sound thump every time. I think we can agree with him and bear in mind that gall-stones are not so common as we have been led to believe in the past. It is not that gall-stones are innocent, but it is the innocent practitioner who has not made the diagnosis of them, and consequently the patient dies, and no greater truism was ever uttered than that very few people with chronic disease die from that disease which they had during life, but from some other complication or secondary disorder. Therefore, the gall-stones have been innocent until their relationship to the disease from which the patient has suffered during life has been considered. It is found that complications are and have been very prominent when it comes to autopsy.

With reference to the remarks by made Dr. Ochsner, I have been traveling with him a good many years, and he has that one thing which I like, and that is, good sound sense. He used to say to me in traveling around together, when somebody made a considerable noise about a surgical condition from which the rest of us did not get the same results, that it was better for us not to call the other fellow a liar because he got better results, or that he juggled his statistics, but to find out the truth, and we learned that such a man was doing better work than we were.

With reference to the Cammidge reaction, we have some assistants who are skilled in that line of work, but I must confess that we have not had good results from the use of this test. While in England I saw Mr. Cammidge and Mr. Robson, and I was satisfied when I got through talking with Mr. Cammidge and seeing his work, that he was able to show things that we had not been able to show, simply because we did not know how to do it. I saw a number of cases operated on by Mr. Robson in which the diagnosis was made by Mr. Cammidge, and I had the opportunity of seeing Cammidge not only make the diagnosis, but make it correctly, and that is an important point. We have started our assistants again in trying this test, and hope to be able to get better results from it.

BOOK REVIEWS

GENERAL SURGERY. A presentation of the scientific principles upon which the practice of modern surgery is based. By Ehrich Lexer, M. D., Professor of Surgery, University of Königsberg. American edition, edited by Arthur Dean Bevan, M. D., Professor and Head of the Department of Surgery, Rush Medical College, in affiliation with the University of Chicago. An authorized translation of the second German edition, by Dean Lewis, M. D., Assistant Professor of Surgery, Rush Medical College. With 449 illustrations in the text, partly in color, and 2 colored plates New York and London: D. Appleton and Co., 1908. xxix and 1041 pages. Cloth, $6.00 net.

"There are certain general principles of pathology and therapeutics and operative technic which apply to all fields of surgery, and when these are thoroughly mastered by the student or practitioner their application to special surgical conditions becomes at once intelligent and easy and scientific." (Editor's preface.) German authors have been foremost in promulgating these principles, and there is no lack of similar works on the subject. Witness — those by Billroth-Winiwarter, Hueter-Lossen, Landerer, Leser, Tillmans, etc. Possibly the bleak shores of the Baltic create an environment which encourages reflection. At any rate, the excellencies of Lexer's "Lehrbuch d. Allgemeine Chirurgie," were so manifest that the first edition was exhausted in a year's time, and the present work is a translation from the second German edition.

The treatise is divided into six parts. Part I (pages 1-142) is devoted to Wounds, Their Treatment and Repair, Aseptic Technic, General and Local Anæsthesia, and General Principles of Plastic Operations. Part II embraces Wound Infections and Surgical Infectious Diseases, and is the longest section in the book (pages 143-484), and is subdivided into, Nature of Infection, Local and General Reaction, Wound Infections Caused by Pyogenic and Putrefactive Bacteria and their Results, Wound Infections of Different Origins, and Surgical Infectious Diseases, e. g., Tetanus, Glanders, Actino- and Botromycosis, and the Infective Granulomata. Necrosis is considered in Part III (pages 485-515).

In Part IV (pages 516-622) we have a very important section — Injuries of Soft Tissues, Bones and Joints, and Their Treatment. This, of course, includes Fractures, Dislocations, Gunshot Wounds, Burns, as well as conditions resulting from such injuries — Collapse, Shock, Fat Embolism, etc. Diseases of the same structures, Nerves, Bones, Joints, etc., are considered in Part V (pages 643-750).

The volume ends with exhaustive sections on Tumors and Cysts (pages 751-1004). There is an Appendix of 11 pages dealing with Crile's Method of Transfusion, and with Opsonic Therapy.

With such a wealth of material, it seems invidious to call attention to any particular section. We believe, however, the chapters in Part II on Infection and Immunity are among the best in the whole work, dealing as they do with these difficult subjects in a peculiarly clear and convincing manner.

The emphasis placed on general surgical principles applicable to diseases in all parts of the body, and the stress laid on the essential pathologic entities, are striking features of the work. The close relationship maintained between pathologic foci with their gross and microscopic manifestations and their surgical treatment is especially commendable.

The distinguished American editor has enriched the work by over 100 pages of text and the same number of illustrations from his surgical clinic. Nearly every page bears witness to the industry with which he has collated the results of his ripe experience in the shape of comments or additions. In places these assume the shape of longer sections from his fellow workers. For example, a chapter of 17 pages on Blastomyocosis by Dr. O. Ormsby.

The difficult task of rendering the German text into idiomatic English has been exceedingly well done, and the magnitude of the undertaking can only be appreciated by those who have essayed similar ones. The publisher's part calls for nothing but praise, the clear print and the numerous and fine illustrations render the perusal of the work a pleasure.

In conclusion the writer would say that no better surgery has been presented to the medical student to form a ground work for his surgical principles, and for the practitioner there is no more scientific, lucid, concise, and practical reference book.

GENERAL PATHOLOGY. By Ernest Ziegler. Translated by Alfred Scott Warthin, Ph. D., M. D. New York: William Wood & Co., 1908.

The appearance of this revised and last edition of Ziegler's General Pathology recalls to mind the death of Prof. Ziegler on the 30th of November, 1905, in the 57th year of his age. His text-book, which has been read throughout the English speaking world, is well known and needs no description as to its general nature, nor recommendation as to its quality. It was fully revised by Prof. Ziegler three years ago, and in it were included the researches upon the etiology of syphilis, other contributions upon tryponosomata, and the newer ideas upon agglutinins, cystotoxins, etc.

This last revision by such an eminent authority, who has watched the development of pathology through three decades, brings to the book a certain air of authority and saneness in decisions which adds immensely to its value. Since it is translated from the German two years after it left Ziegler's hands, an opportunity was given Prof. Warthin to add many of the recent observations upon the effects of Rœntgen irradiation, phagocytes, opsonins, blood plates, thrombosis, regeneration, relapsing fever, protozoa, etc.

The book is a volume of 750 pages with over 600 illustrations and attempts to present in as concise a manner as possible, the various essential facts concerning general pathology. In addition the bibliography is complete, especially as to German literature.

MODERN CLINICAL MEDICINE. DISEASES OF THE NERVOUS SYSTEM. Edited by Archibald Church, M. D. An authorized translation from Die Deutsche Klinik, under the editorial supervision of Julius L. Salinger, M. D. D. Appleton & Co., 1908.

This rather large volume of 1,200 pages is devoted to the discussion of thirty-two subjects of major neurologic importance by as many different authors. Regardless of what the publishers intended it to be, it is more than a text-book, yet less than a series of monographs. To those familiar with the articles as they appeared in their original Deutsche Klinik form, the text appears to have lost some in the English translation. This is to be expected when we consider that aside from the complexities in German composition and syntax each contributor reflects a style and mode of expression peculiarly his own. The subject matter of the first two chapters by Rothman and Rosin, on the Anatomy and Histopathology of the Central Nervous System, is well presented and amplified by very good illustrations in black and white. The chapter written on General Neurologic Diagnosis by Schuster is not satisfying in all its parts, nor is the English used free from objection. For instance, on page 203, in discussing the Babinski toe-sign, we read "on application of a mild irritant to the sole of the foot," meaning, of course, gentle stroking of the plantar surface.

Quincke's article on lumbar puncture is in every respect adequate, comprehensive, and a model of its kind. Not the least valuable part of this chapter is the inclusion of a full bibliography of the recent and more important contributions to this subject.

Wernicke has written on aphasia, but the controversy now pending in regard to the views and theories expressed by Marie have been omitted.

The articles by Erb on tabes dorsalis and paralysis agitans are classic and suffer less in the translation than any others in the book. Lüthje, Bernhardt and Kassierer have contributed chapters that are clear and compact on hereditary ataxia, peripheral nerve paralyses and polyneuritis.

The article on "The Present Status of Graves' Disease" by Eulenberg will appeal to those English readers who are the proponents of the view that Basedow's disease is a medical disease and only in the exceptional instances amenable to surgical intervention.

It would transcend the limits of a brief review to mention all the contributions in detail. Some are good and others so incomplete as to suggest their having been prepared without that high regard for merit that distinguishes nearly all of the book values coming to us from German sources in medicine. D. O. H.

BOOKS RECEIVED

A MANUAL OF OBSTETRICAL TECHNIQUE. By Joseph Brown Cooke, M. D. Philadelphia: J. B. Lippincott Company, 1908.

UBER APPENDICITIS WAHREND SCHWANGERSCHAFT UND GEBURT. Von Gerhard Renvall. Berlin, 1908, Verlag von S. Karger, Karlstrasse 15.

DIE LEHRE VON DER KONGENITALEN TUBERKULOSE. Mit besonderer BERÜCKSICHTINUNG DER PLACENTARTUBERKULOSE. Von Dr. Anton Sitzenfrey. Berlin, 1908, Verlag Von S. Karger.

DIE PLACENTA MARGINATA, UND IHRE ENTSTEHUNG. Von Dr. P. Sfameni. Berlin, 1908. Verlag Von S. Karger.

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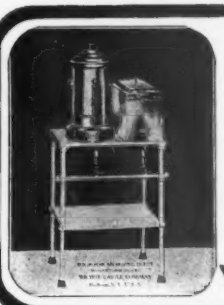

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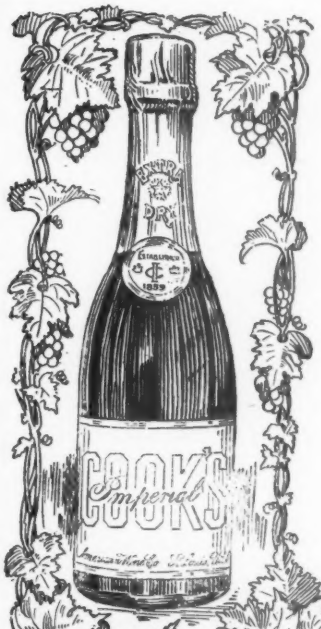

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The Combined Operation for Carcinoma of the Rectum. . . . . Jos. A. BLAKE, M. D., New York City

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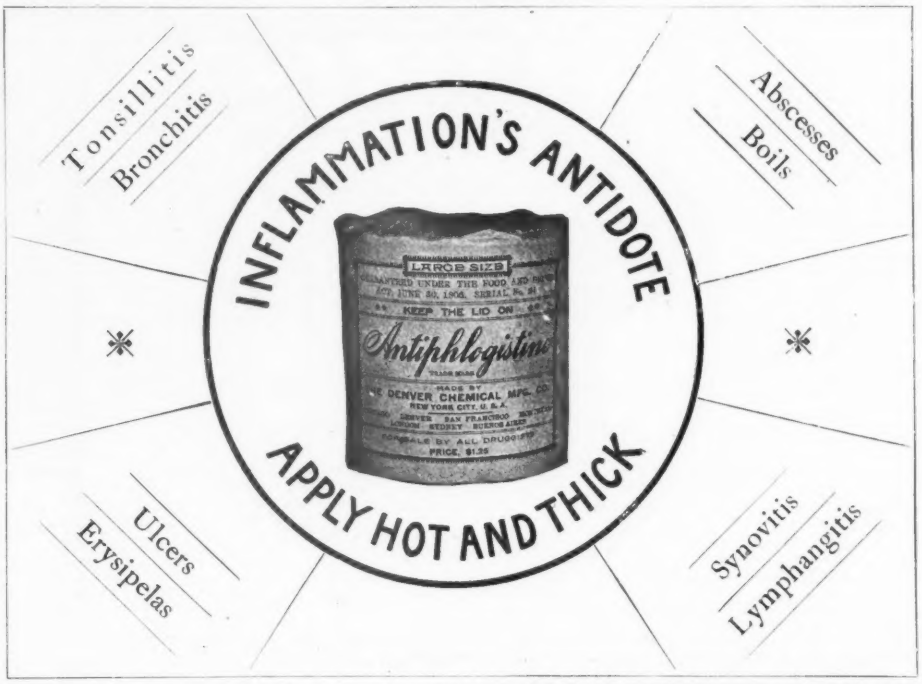

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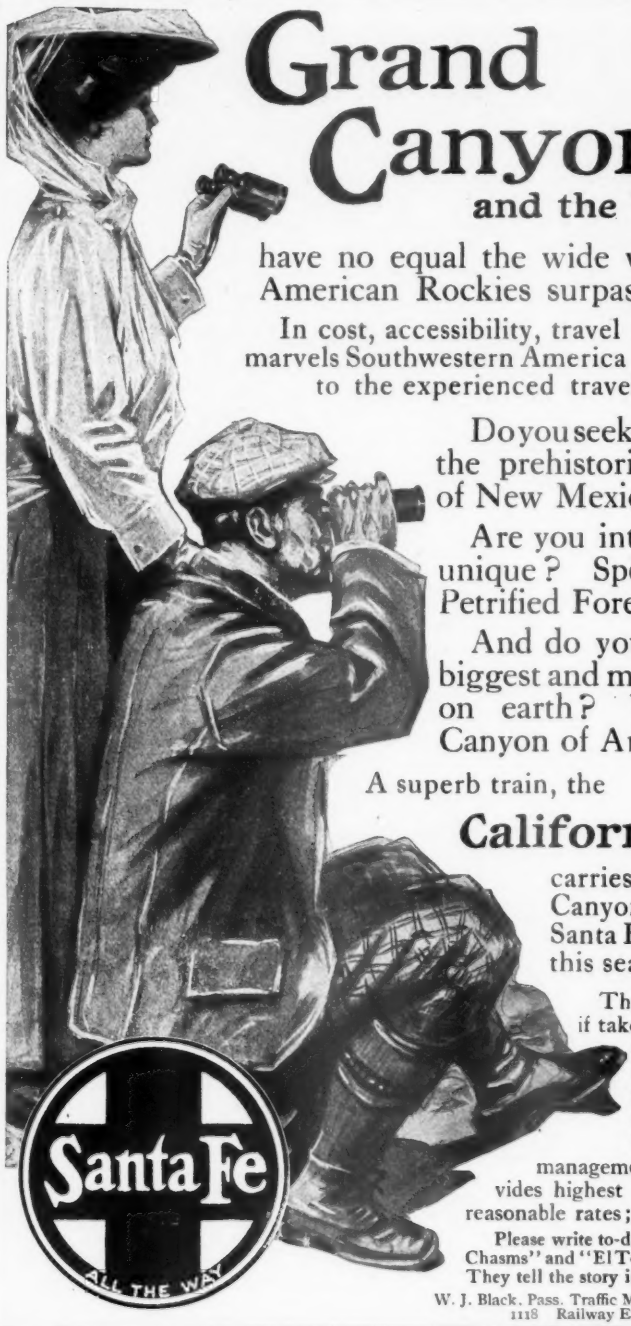

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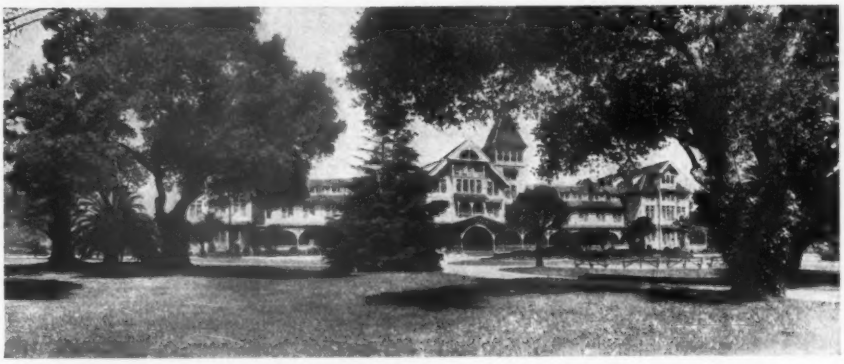

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